GASTRO ESOPHAGEAL REFLUX DISEASE AND OBESITY

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CERTIFICATE

This is to certify that the dissertation titled **"Gastro Esophageal Reflux Disease and Obesity"** is the bonafide original work of **Dr. MALA MAHALAKSHMI, J.** in partial fulfillment of the requirements for M.D. Branch – I (General Medicine) Examination of the Tamilnadu DR. M.G.R Medical University to be held in MARCH 2009. The period of study was from July 2007 to July 2008.

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DECLARATION

I, **Dr. MALA MAHALAKSHMI. J**, hereby solemnly declare that the dissertation titled **"GASTROESOPHAGEAL REFLUX DISEASE AND OBESITY"** was done by me at Government Stanley Medical College and hospital during 2007-2008 under the guidance and supervision of my unit chief Prof. S. SUNDAR.

The dissertation is submitted to the Tamilnadu Dr.M.G.R. Medical University towards the partial fulfillment of requirement for the award of M.D degree (Branch-1) in General Medicine.

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CONTENTS

INTRODUCTION

Gastroesophageal reflux disease is multifactorial process and a common problem which accounts for a sizeable proportion in terms of health care costs to diagnose and treat the condition[1].

DEFINITION

The reflux of acid, particularly after meals, is a physiologic process, the simple presence of gastroesophageal reflux (GER) or occasional symptoms of heartburn or acid regurgitation cannot be defined as a disease.

GERD is the failure of normal antireflux barrier to protect against frequent and abnormal amounts of gastric contents moving retrograde effortlessly from the stomach into the esophagus. A globally acceptable Montreal definition and classification of GERD can be applied in clinical practice and in research[2]. This international group defined GERD as "a condition which develops when the reflux of stomach contents causes troublesome symptoms and/or complications." Troublesome symptoms are defined by the patient to affect their quality of life. Mild symptoms occurring 2 or more days per week or moderate to severe symptoms occurring more than 1 day per week are often considered troublesome by patients. Patients may be diagnosed based on typical symptoms alone or on tests demonstrating reflux of stomach contents (e.g. pH testing, impedance monitoring) or the injurious effects of the refluxate (endoscopy, histology, electron microscopy), in the presence of typical or atypical symptoms or complications. This new definition

also recognizes that the refluxate causing symptoms may be weakly acidic or gaseous.

The group further divided the manifestations of GERD into esophageal and extraesophageal syndromes, with extraesophageal syndromes divided into established and proposed associations. In primary care, most patients are initially uninvestigated and present with symptomatic syndromes, either typical reflux complaints of heartburn and regurgitation or reflux-related chest pain. After investigation, usually endoscopy with histology, patients can be further classified as having the “syndromes with mucosal injury” to include reflux esophagitis, stricture, Barrett esophagus, or esophageal adenocarcinoma. The definition thus allows symptoms to define the disease but permits further characterization if mucosal injury is found. This group also recognized laryngitis, cough, asthma, and dental erosions as possible extraesophageal syndromes of GERD. The statement was restrained in defining a causal relationship, however, because of the lack of high-level evidence, especially showing a beneficial effect of reflux treatments on the extraesophageal syndromes and the reality that these syndromes are usually multifactorial, with GERD as one of several potential aggravating cofactors.

EPIDEMIOLOGY

The incidence of GERD, defined as at least weekly heartburn and/or acid regurgitation, in the Western world generally ranges between 15% and 25%, whereas in Asia the prevalence is reported to be less than 5%[3,4]. Time trends confirm a significant increase in the prevalence of reflux symptoms

averaging 5% annually in North America, 27% annually in Europe, and only 1% in Asia possibly because of lower dietary fat, lower BMI and low acid output due to H.pylori infection among Asians[4] although the scenario is changing on account of changing diet habits, increasing BMI and H.pylori eradication among Asians. The disease is a relapsing and remitting disorder, but in contrast to the data for period prevalence, there are few longitudinal studies that describe the incidence of heartburn in the population. Based on only two studies from the Western world, the incidence of GERD can be estimated at 5 per 1000 person-years or an adjusted yearly incidence of weekly heartburn of approximately 1.5% to 3%.

Even less is known about the prevalence of reflux esophagitis. A population-based endoscopic study suggests that asymptomatic esophagitis is common. In a random sample of a Swedish adult population, reflux symptoms were reported by 40% and esophagitis was diagnosed in nearly 16%. One third of those who had esophagitis, however, had no symptoms of GERD[5]. Two other population-based studies found the prevalence of esophagitis to be nearly 12% in Italy but only 7% in Japan[4].

RISK FACTORS

Age: The effect of increasing age on the prevalence of GERD symptoms is unclear. European studies report a slight but significant association, but the relationship was not observed for heartburn with or without acid regurgitation[6]. A recent study suggested an association between advancing age and milder reflux symptoms but more severe esophagitis [7].

Gender: All studies report a similar prevalence of heartburn in men and women[5]. On the other hand, endoscopy database studies find male sex a significant risk factor for reflux esophagitis[8].

BMI: Cross-sectional studies and systematic reviews consistently find that obesity is associated with a statistically significant increase in the risk for reflux symptoms, erosive esophagitis, Barrett esophagus, and esophageal adenocarcinoma[9,10,11]. In these studies, obesity (BMI >25) was associated with 2.5- to 3.0-fold increase in these presentations of GERD.

Helicobacter Pylori Infection: an environmental factor that has declined as GERD, Barrett esophagus, and esophageal adenocarcinoma have increased in developed countries[12]. A systematic review of observational studies has confirmed that there is a negative association between H pylori and GERD, although this finding is most apparent in Asian countries[13]. The causative mechanism for this protective effect is the H pylori-induced gastritis, involving the antrum and corpus, which decreases the parietal cell mass, reduces acid secretion, and elevates gastric pH[14].

Genetics: There have been two studies[15,16] assessing the prevalence of reflux symptoms in monozygotic versus dizygotic twins. Data from the Swedish Twin Registry[15] suggested that 31% (95% CI, 23%–39%) of GERD is caused by additive genetic factors, whereas a United Kingdom Twin Registry study[16] reported that this value was 43% (32%–55%). Although one group defined a

locus on chromosome 13 associated with severe pediatric GERD[17], this has not been confirmed in adults. The genetic mechanisms are unknown but may be related to a smooth muscle disorder associated with hiatal hernia, low LES pressure, and impaired esophageal motility

PATHOGENESIS: ROLE OF OBESITY

GERD results from an imbalance between defensive factors protecting the esophagus (antireflux barriers,esophageal acid clearance,tissue resistance) and aggressive factors from the stomach (gastric acidity,volume and duodenal contents). Parallel increase in prevalence of GERD and obesity in the West led to studying of obesity as a contributing factor[95]. Obesity may alter the anatomy of the antireflux barrier in the following ways:

- Pressure and integrity of the diaphragmatic crura which prevents
- ` reflux during sneezing,coughing,bending is compromised
- Lax phrenoesophageal ligaments
- Change in the acute angle of His
- Hiatus hernia associated with abdominal obesity alters the intra abdominal location of lower esophageal sphincter,causes lower basal LES pressure[96], eliminates the increase in LES pressure during straining, increases the transient LES relaxations during gastric distention,[97,98] impairs esophageal acid clearance[99] and alters esophagogastric junction compliance[100]

CLINICAL PRESENTATIONS

Heartburn and acid regurgitation are the classic symptoms of GERD. Heartburn describes a burning feeling, rising from the stomach or lower chest and radiating toward the neck, throat, and occasionally, the back[18]. It occurs postprandially, particularly after large meals or after eating spicy foods, citrus products, fats, chocolates, or drinking alcohol. The supine position or bending over may exacerbate heartburn. Night time heartburn may cause sleeping difficulties and impair next-day function[19]. The frequency and severity of heartburn does not predict the degree of esophageal damage[7]. The effortless regurgitation of acidic fluid, especially after meals and worsened by stooping or the supine position is suggestive of GERD. Among patients who have daily regurgitation LES pressure is usually low, many have associated gastroparesis, and esophagitis is common, making this symptom more difficult to treat than classic heartburn. Symptoms such as dysphagia, odynophagia, globus sensation, burping, water brash, and cough are other possible presentations of GERD, but their diagnostic yield is uncertain. Odynophagia may be seen with severe reflux esophagitis, but usually suggests an infection or pill-related esophagitis. Water brash is the sudden appearance in the mouth of a salty fluid. It is not regurgitated fluid, but rather secretions from the salivary glands in response to acid reflux[20].

The clinical accuracy of heartburn or regurgitation in diagnosing GERD is difficult to define. A recent systematic review[21] identified seven studies that assessed the accuracy of these reflux symptoms in diagnosing GERD in more than 5000 patients. Endoscopy with the presence of esophagitis has excellent specificity; thus, it was used as the gold standard to assess the sensitivity of heartburn and regurgitation. Unfortunately the sensitivity of these classic reflux symptoms was poor, with a range of 30% to 76% and a pooled sensitivity of 55% (95% CI, 45% – 68%). Many patients who have atypical upper gastrointestinal (GI) symptoms thus may have GERD.

Some patients who have GERD are asymptomatic. This is particularly true in elderly patients, perhaps because of reduced gastric acidity from chronic H pylori infection or decreased pain perception. Many elderly patients present first with complications of GERD because of long-standing disease with minimal complaints. For example, up to one third of patients who have Barrett esophagus are insensitive to acid at the time of presentation[22].

DIAGNOSTIC TESTS

A large number of tests are available for evaluating patients who have suspected GERD. Many times these tests are unnecessary, because the presence of frequent heartburn and acid regurgitation are sufficiently accurate to identify the disease and begin medical treatment. This is not always the case, however, and clinicians must decide which tests to choose so as to make the diagnosis in a reliable, timely, and cost-effective manner, depending on the information desired (Table 1)[23].

Diagnostic tests for gastroesophageal reflux disease

Tests for reflux

- Intraesophageal pH monitoring
- Ambulatory bilirubin monitoring (bile reflux)
- Ambulatory impedance and pH monitoring (non-acid reflux)
- Barium esophagram

Tests to assess esophageal mucosal damage

- Endoscopy
- Esophageal mucosal biopsy
- Barium esophagram

Tests to assess symptoms

- Empirical trial of PPIs
- Intraesophageal pH monitoring with symptom analysis

Tests to assess esophageal function

- Esophageal manometry
- Esophageal impedance
- Barium esophagram with fluoroscopy

Upper Endoscopy

The identification of esophagitis at the time of endoscopy is highly specific (90%–95%) for GERD[24] but has a sensitivity of only approximately 50%. Multiple classification systems for esophagitis have been proposed, some

are confusing, and none have worldwide acceptance[25]. The most thoroughly evaluated esophagitis classification is the Los Angeles (LA) system, which is gaining acceptance in the United States and Europe (Fig. 3)[26]. In referral centers, approximately 50% of patients have esophagitis, but in primary care and the general population, the rate of esophagitis is more in the range of 10% to 30%[4]. Most patients who have esophagitis have mild LA grade A-B disease and only 10% have the more severe LA grade C-D esophagitis[27]. Endoscopy can also evaluate complications of GERD, such as peptic strictures and Barrett esophagus and is recommended if patients have alarm symptoms, such as progressive dysphagia, weight loss, or iron deficiency anemia[28]. In routine clinical practice, endoscopy is reserved for evaluating patients who have alarm symptoms, for suspected GERD complications, and for surveillance for Barrett esophagus in patients who have chronic reflux complaints[29].

Biopsy: Over the years esophageal biopsies have had a varying role in the evaluation of GERD. The presence of eosinophils (<15 per high powered field) and markers of increased epithelial turnover (basal cell hyperplasia and prolongation of rete peg) have reasonable sensitivity but poor specificity, whereas neutrophils in the esophageal mucosa are specific but not sensitive[30]. Electron microscopy of esophageal biopsies suggests that dilated intercellular spaces could be an early marker of mucosal injury, whereas the endoscopy still seems normal (Fig. 4)[31,32]. Several studies consistently find the intercellular spaces at least two to three times greater in patients who have erosive and nonerosive GERD compared with healthy control subjects[32]. Aggressive acid

suppression therapy seems to normalize the width of the intercellular spaces[32]. Unfortunately these spaces are more difficult to define by light microscopy. In clinical practice, biopsies are usually not taken in patients who have classic reflux esophagitis unless necessary to exclude neoplasm, infection, pill injury, bullous skin disease, or eosinophilic esophagitis (>20 eosinophils per HPF). The current primary indication for esophageal biopsies is to determine the presence of Barrett epithelium[29]. When this diagnosis is suspected, biopsies are mandatory and best done when esophagitis is healed.

Esophageal pH Monitoring

Ambulatory intraesophageal pH monitoring is the standard for establishing pathologic reflux[23]. Traditionally the pH probe is passed nasally, positioned 5 cm above the manometrically determined LES, and connected to a battery-powered data logger capable of collecting pH values every 4 to 6 seconds for 24 hours. Patients record meals, sleeping, and when symptoms are experienced. Acid reflux episodes are defined as a pH drop of less than 4. The total percent of time the pH is less than 4 is the most reproducible measure of GERD, with the reported upper limits of normal ranging from 4% to 5.5%[33]. The sensitivity of 24-hour pH monitoring in patients who have esophagitis approaches 90% with a specificity of 85% to 100%. In patients who have normal endoscopy in which pH testing may be most needed, the sensitivity is only 60% and the specificity from 85% to 90%[34]. Clinical indications for ambulatory pH monitoring include (1) before fundoplication to insure the presence of pathologic reflux in patients who have a normal endoscopy,

(2) after antireflux surgery if heartburn symptoms persist, (3) patients who have reflux symptoms and a normal endoscopy not responding to PPI treatment, and (4) patients who have suspected extraesophageal symptoms of GERD[34].

Two new advances are improving the role of pH testing in evaluating GERD. The first is a wireless device (Bravo pH probe, Medtronics, Minneapolis, MN) the size of a vitamin pill attached, usually by endoscopy, 6 cm above the Z-line (Fig. 5)[35]. This decreases patient discomfort, allows for longer (48 hours or more) monitoring, and may increase test sensitivity by allowing patients to more comfortably carry out their usual activities[36]. The capsule detaches and passes spontaneously within 2 weeks. Rare patients may note severe pain after attachment, which resolves spontaneously with endoscopic removal. Two significant complications have occurred with this device—one report of esophageal bleeding requiring transfusion and one esophageal perforation[37].

The second technologic advancement combines multichannel intraluminal impedance monitoring with pH sensors to detect acid, weak acid, and non-acid reflux using a transnasal catheter over 24 hours[38]. The number of respective reflux episodes, rather than percentage of exposure time, is the critical measurement, with normal values established from United States and European studies[38]. The results of several studies suggest that impedance-pH monitoring is useful in the evaluation of patients who have PPI-resistant typical reflux symptoms, especially regurgitation complaints, and chronic unexplained cough[39,40,41].

Barium Esophagram

The barium esophagram is inexpensive and less invasive than endoscopy. It is most useful in demonstrating strictures, rings, hiatus hernias, and major abnormalities in peristalsis. The barium esophagram's ability to detect esophagitis varies, with sensitivities of 79% to 100% for moderate to severe esophagitis, whereas mild esophagitis is usually missed[23]. It is also not reliable for detecting Barrett esophagus.

Esophageal Manometry

Esophageal manometry allows assessment of LES pressure and relaxation and peristaltic activity, including contraction amplitude, duration, and velocity. It is generally not indicated in the evaluation of patients who have uncomplicated GERD, however, because most have normal resting LES pressure[42]. Esophageal manometry to document adequate esophageal peristalsis is traditionally recommended before antireflux surgery. If the study identifies ineffective peristalsis (low amplitude or frequent failed peristalsis), then a complete fundoplication may be contraindicated. This assumption has recently been challenged, however, by several studies finding that reflux control was better and dysphagia no more common in patients who had weak peristalsis after a complete as opposed to partial fundoplication[43]. An improvement of traditional manometry, combining it with impedance testing, is helping to clarify this controversy. Using this technology, a recent study found

that less than 50% of patients who had ineffective peristalsis had a significant delay in esophageal bolus transit measured by impedance[44].

Proton Pump Inhibitor Test

An empiric trial of acid suppression is the simplest method for diagnosing GERD and assessing its relationship to symptoms. With the advent of PPIs, this test has become the first diagnostic study used in patients who have classic or atypical reflux symptoms without alarming complaints. Symptoms usually respond to a PPI test in 1 to 2 weeks. If symptoms disappear with therapy and then return when medication is discontinued, GERD is established. A systematic review[45] identified 15 studies that assessed the accuracy of normal or high dose PPIs for 1 to 4 weeks in the diagnosis of GERD. The pooled sensitivity was good at 78% (95% CI, 66%–86%), but the specificity was poor at 54% (95% CI, 44%–65%) when 24-hour ambulatory pH was used as a gold standard. Nevertheless an empiric PPI trial for diagnosing. GERD offers many advantages: the test is office-based, easily done, inexpensive (especially with over-the-counter PPIs), available to all physicians, and may avoid needless procedures. For example, one study showed a savings of greater than $570 per average patient because of a 59% reduction in the number of diagnostic tests (upper endoscopy, pH tests)[46]. Disadvantages are few, but include a placebo response and uncertain symptomatic endpoint if symptoms do not totally resolve with extended treatment.

Complications

There are few data on the long-term outcome of patients who have varying severities of GERD. Severity and duration of symptoms seem to have a poor correlation with the presence or severity of esophagitis[7]. Furthermore, there is some controversy whether GERD exists as a spectrum of disease severity or as a categoric disease in three distinct groups: nonerosive and erosive reflux disease and Barrett esophagus[24,47]. The recent European ProGERD study involving nearly 4000 patients sheds some light on the progression or regression of GERD over 2 years[47]. After endoscopy to assess the presence or absence of esophagitis, all patients were treated with 4 to 8 weeks of esomeprazole then returned to their primary care physician. Two years later all patients underwent a second endoscopy with biopsies. As shown in Fig. 6, after 2 years 25% of patients who had nonerosive GERD progressed to LA grade A-B esophagitis but severe LA grade C-D esophagitis was rare (<1%). Likewise, only 1.6% of LA grade A-B patients progressed to severe disease, whereas most (61%) regressed to nonerosive disease. Even the severe LA grade C-D patients had a good prognosis, with 42% regressing to milder esophagitis and 50% regressing to a nonerosive state. Patients who had LA grade C-D esophagitis were at a greater risk 2 years later for developing Barrett esophagus: 5.8% compared with 1.4% for LA grade A-B and 0.5% for nonerosive disease. These data suggest GERD more likely is a spectrum of disease that tends to regress in severity after it comes to medical attention,

regardless of treatment. The progression of Barrett esophagus may be an artifact of better detection after esophagitis has healed[48].

Peptic Esophageal Strictures

Esophageal strictures have a prevalence of approximately 0.1% and are associated with white race, male gender, and increasing age[49]. Patients usually present with dysphagia for solids, but unlike malignant strictures, weight loss is uncommon because their appetite is good. As dysphagia progresses, heartburn often decreases, reflecting the stricture acting as a barrier to further reflux. Peptic strictures are smooth walled, tapered, circumferential narrowing in the lower esophagus that are usually less than 1 cm long. A mid to upper esophageal stricture should raise concern about Barrett esophagus or malignancy. Although once controversial, today a Schatzki ring is considered a forme fruste of an early peptic stricture[50]. All stricture patients should undergo endoscopy, at least initially, to confirm the benign nature of the disease and, if necessary, to take biopsies to exclude cancer and Barrett esophagus. Symptomatic patients can be dilated by various bougies[51]. Dysphagia relief generally occurs when the lumen is greater than 15 mm and associated esophagitis has healed[52].

Barrett Esophagus

Barrett esophagus is the consequence of severe GERD in which the squamous epithelium of the distal esophagus is replaced by specialized columnar mucosa containing goblet cells (intestinal metaplasia). The disease is more common in white men, rare before age 50 years, and present in 1% to 2%

of patients referred for endoscopy over this age threshold[8]. Bile reflux and obesity have been associated with an increased risk for Barrett esophagus[9,53]. The diagnosis can be suspected at endoscopy and its circumferential involvement and maximum proximal extension described using the new Prague classification[54]. Histology is required, however, to confirm the diagnosis and to define the potentially premalignant intestinal metaplasia. Detection of Barrett esophagus is highest after suspected patients have been on PPIs for 8 to 12 weeks[48]. In the era of PPIs, Barrett esophagus is easy to treat and only of major interest because of an increased risk for developing esophageal adenocarcinoma, estimated at between 0.5% and 1% each year[55]. Increased duration, frequency, and severity of reflux symptoms have been shown to be risk factors for this cancer[56,57,58].

Extraesophageal Manifestations

Gastroesophageal reflux may be the cause of a wide spectrum of conditions, including non-cardiac chest pain, asthma, posterior laryngitis, chronic cough, recurrent pneumonitis, and even dental erosion[59]. GERD-related chest pain may mimic angina pectoris, even to the point of being induced by exercise. Most of these patients also have heartburn symptoms[60]. The mechanism of the pain is poorly understood, likely because of the volume and duration of acid reflux, secondary esophageal spasm, or prolonged contractions of the longitudinal muscle[61]. The causal link between GERD and pulmonary and ear, nose, and throat complaints is much more circumspect[59]. Although the possible mechanisms from animal studies are plausible

(i.e., microaspiration and vagal reflex), most suspected patients are heartburn-free and do not have esophagitis, hiatus hernia, or low LES pressure. Unfortunately pH testing (distal or proximal), although frequently abnormal in these patients, does not predict their response to medical or surgical therapies.

Medical Treatment

Lifestyle and Over-the-Counter Medications:

Numerous dietary and lifestyle modifications are advocated for the treatment of GERD. They are frequently first-line therapy for patients who have mild disease and often adjunct therapy even for patients on PPIs. In a recent evidence-based review, studies of smoking, alcohol, chocolate, fatty foods, and citrus products showed physiologic evidence that their intake can adversely affect symptoms or esophageal pH. There was little evidence, however, that cessation of these products predictably improved GERD symptoms. Only elevation of the head of the bed, left lateral decubitus positioning, and weight loss was associated with GERD improvement in case-controlled studies[62].

Over-the-counter (OTC) antacids and H2RAs are useful in treating mild and infrequent heartburn symptoms, especially when symptoms are brought on by lifestyle indiscretions. In one recent meta-analysis[63], the relative benefit increase compared with the overall placebo response was up to 41% with H2RAs, 60% with alginates, and 11% with antacids. Antacids rapidly relieve heartburn symptoms, the major reason these drugs are so popular for mild, intermittent symptoms. Although their onset of relief is not as rapid as antacids,

OTC H2RAs have a longer duration of action, up to 6 to 10 hours. From a practical standpoint, they are most useful when taken before a potentially refluxogenic activity, such as a heavy meal, eating late at night, or exercise.

Proton Pump Inhibitors

PPIs revolutionized the treatment of GERD and currently are the mainstay of acute and maintenance treatment regimens. This class of drugs markedly diminishes gastric acid secretion over a 24-hour period by inhibiting the final common pathway of acid secretion, the H+K+ ATPase pump. Their superior efficacy compared with H2RAs is based on their ability to maintain an intragastric pH of less than 4 between 15 and 21 hours, compared with approximately 8 hours daily with H2RAs.

In a recent Cochrane review[64], PPIs were more effective than placebo in healing esophagitis (RR = 0.23; 95% CI, 0.01–0.05) with a number to treat (NNT) of 2 (95% CI, 1.4–2.5). The review also identified 26 trials involving 4064 patients that compared PPIs with H2RAs. PPIs were superior to H2RAs in healing esophagitis at 4 to 6 weeks (RR = 0.47; 95% CI, 0.41–0.53) with an NNT of 3 (95% CI, 2.8–3.6). Another Cochrane systematic review found that PPI therapy was superior to placebo and H2RAs in endoscopy-negative GERD and undiagnosed reflux symptoms in primary care, although the effect was not as marked as with esophagitis[65]. Cochrane reviews also have identified the superiority of PPIs over H2RAs in maintaining the remission of esophagitis over 6 to 12 months[66]. Among 10 randomized trials, the relapse rate for

sophagitis was 22% on PPIs compared with 58% on H2RAs, with an NNT of 2.5 (95% CI, 2.0–3.4).

Until recently the therapeutic efficacy between PPIs was similar. Recent large randomized controled trial (1000–2500 patients), however, have found the newest PPI, esomeprazole 40 mg, superior to omeprazole 20 mg and lansoprazole 30 mg in healing esophagitis[67]. The therapeutic advantage is minimal with mild LA grade A-B esophagitis (NNT 50 and 33, respectively) and greatest with severe LA grade C-D esophagitis (NNT 10 and 8, respectively). This superiority is related to higher systemic bioavailability and less inter-patient variability with esomeprazole.

Treatment Of Complicated Gastroesophageal Reflux Disease And Its Extraesophageal Presentations

The extensive use of PPIs has markedly affected treatment of peptic strictures and esophageal rings. Several studies note an approximate 33% decline in the incidence of recurrent strictures. The timeline for this decrease parallels the marked increase in PPI use since 1995 (Fig. 7)[68]. Another study convincingly shows that in patients who have symptomatic Schatzki rings, maintenance PPI therapy after bougienage markedly decreases future relapses of the rings[69]. In a randomized study, 30 patients who had symptomatic rings without esophagitis were dilated and randomized to placebo or omeprazole 20 mg per day. In the treated group, one patient relapsed after 13 months, whereas seven patients relapsed on placebo after a mean of 20 months.

The efficacy of PPI treatment in the extraesophageal presentations of GERD is more variable. There are two systemic reviews[70,71], both suggesting that patients who have non-cardiac chest pain respond to PPIs better than to placebo. These reports identified eight RCTs that assessed 321 patients who had a pooled relative risk for continued chest pain after PPI therapy, compared with placebo of 0.54 (95% CI, 0.41–0.71), with an NNT of 3 (95% CI, 2–4). Systemic reviews, however, do not support the efficacy of aggressive acid suppression, particularly with PPIs in other extraesophageal disorders, such as chronic cough[72], asthma[73], or ear, nose, and throat disorders[74].

Sleep disturbances may occur in up to 75% of patients who have GERD, impairing quality of life. In a large multicenter study, patients who had GERD-associated sleep disturbances and nighttime heartburn were randomized to two doses of esomeprazole (40 mg and 20 mg) or placebo for 4 weeks[75]. GERD-related sleep disturbances resolved in significantly more patients on esomeprazole 40 mg (73.7%) or 20 mg (73.2%) than those who received placebo (41.2%). These changes were associated with improved sleep quality and daytime productivity.

Refractory Gastroesophageal Reflux Disease

Traditionally patients who have reflux symptoms no longer undergo initial endoscopy, but rather are given a 4- to 8-week trial of a PPI. Failure to improve occurs in 25% to 42% of patients, thus placing them in a more difficult to manage group. At this point the physician should insure patient compliance and review timing of the PPI dose (1/2 to 1 hour before meals).

One recent study found that nearly 70% of primary physicians and 20% of gastroenterologists gave the PPI at bedtime or did not believe the relationship to meals was important[76]. Switching to a second generation PPI (i.e., pantoprazole, esomeprazole) may be a reasonable alternative. This was recently confirmed in a multicenter study of patients who had persistent heartburn symptoms while receiving lansoprazole 30 mg once daily[77]. Switching to a single dose of esomeprazole (40 mg) was as effective as twice daily lansoprazole in relieving heartburn complaints over 8 weeks of therapy. Most physicians, however, increase the current PPI to twice daily dosing (before breakfast and dinner), with up to 25% of patients responding[78].

Those patients doing no better fall into the "refractory GERD" category[79]. The critical diagnostic test is upper GI endoscopy, which identifies patients who have esophagitis or no esophagitis. The largest percentage of these patients have refractory symptoms with no esophagitis. These patients may require 24-hour pH testing on PPI therapy, impedance testing, and consideration of other diagnoses, such as achalasia, gastroparesis, and functional heartburn.

Safety Concerns

Initial concerns about PPIs causing gastric malignancies in rats have not been substantiated in other animal models or long-term patient studies. Fundic gland polyps are the most common gastric polyp found at endoscopy. Their association with chronic PPI use has been a topic of debate since these drugs were first described. A recent study evaluated 599 patients of whom 322 used

PPIs and 107 had fundic gland polyps[80]. Long-term PPI use was associated with up to a fourfold increase in the risk for fundic gland polyps. Low-grade dysplasia was found in one fundic gland polyp. Etiologically these polyps seem to arise because of parietal cell hyperplasia and parietal cell protrusions resulting from acid suppression.

Recent studies confirm that chronic acid suppression may be associated with an increased risk for community-acquired pneumonias and enteric infections. In a large Scandinavian population-based study[81], the adjusted relative risk for pneumonia among current PPI users compared with those who stopped using PPIs was 1.89 (95% CI, 1.36–2.62). Current users of H2RAs had a 1.63-fold increased risk for pneumonia (95% CI, 1.07–2.48) compared with those who stopped. A significant positive dose–response relationship was observed in the PPI users. Likewise a recent systematic review found an increased risk for enteric infections with acid suppression[82]. The correlation was stronger with Salmonella, Campylobacter, and other enteric infections compared with Clostridium difficile, and greater with PPI compared with H2RA therapy.

PPIs also may alter calcium metabolism through induction of hypochlorhydria interfering in insoluble calcium absorption or through reduced bone resorption through inhibition of osteoclastic vacuolar proton pumps. In a recent nested case-control study[83]. the risk for hip fractures was significantly increased among patients prescribed more than 1 year of PPI therapy (OR, 1.44; CI, 1.30–1.59) and among those on long-term, high-dose PPIs

(OR, 2.65; 95% CI, 1.80–3.90; P<.001). The strength of the association increased with increasing duration of PPI therapy. For elderly patients requiring long-term PPIs, it may be prudent to re-emphasize increased calcium intake, preferably from a dairy source, and co-ingestion of a meal when taking insoluble calcium supplements.

New Drug Treatments

New drug treatments have primarily targeted transient LES relaxation, the common motility abnormality in all forms of GERD. Several agents, including cholecystokinin A agonists, anticholinergic drugs, nitric oxide synthase inhibitors, morphine, cannaboid, and gamma-aminobutyric acid B (GABA) agonists have been shown to reduce transient LES relaxation and episodes of acid reflux[84]. The only agent available for oral therapy is baclofen, a GABA agonist. Several studies show that 10 to 20 mg of baclofen three to four times daily for up to 4 weeks reduces 24-hour esophageal acid and bilirubin reflux[85,86]. Baclofen needs to be titrated upward slowly, beginning at 5 mg daily and increased over 10 days to 40 to 60 mg per day. Side effects are common and include drowsiness, nausea, and the lowering of the threshold for seizures. New compounds with more specific and better targeted action need to be developed. Another approach has been to develop newer rapid-acting PPIs, such as potassium-competitive acid blockers (P-CAB). Unlike the traditional PPIs that bind irreversibly to the proton pump, this new class of compounds blocks acid secretion by way of potassium-competitive inhibition of the H+K+ ATPase. This results in rapid onset with almost complete acid blockade

achieved within 30 minutes of administration[84]. Unfortunately recent phase III studies found the P-CABs no more effective than esomeprazole in the rapid relief of heartburn symptoms.

Endoscopic Treatment

Various endoscopic techniques for the treatment of GERD have been developed as alternatives to antisecretory therapy or antireflux surgery[87]. These techniques include the delivery of radiofrequency energy to the gastroesophageal junction, injection of bulking agents , or implantation of a bioprosthesis into the LES, and suture plication of the proximal gastric folds (Endoscopic Plication System). Studies to date have primarily enrolled PPI-dependent patients who do not have severe esophagitis or large hiatus hernia. Each of these techniques decreases reflux symptoms, improves quality of life, and decreases the need for antisecretory medications. Physiologic studies, however, are much less impressive, with LES pressure rarely increasing, pH normalizing in only 30% of patients, and even mild esophagitis infrequently healing. Sham studies show a decrease in heartburn symptoms and improved quality of life after the active therapy compared with the sham group after 3 to 6 months[87]. Only the Plication study showed a significant decrease in pH values, by only 18%, whereas no change in pH or LES parameters was observed in studies using the other techniques[88].

Most studies of endoscopic therapy have only limited follow-up information on a small number of patients. The durability of these techniques beyond 1 to 2 years remains unclear and seems to gradually decrease over time.

The cost-effectiveness of these techniques is difficult to define. Most important, safety issues have haunted these procedures, especially when used in the broader community of gastroenterologists. Chest pain, bleeding, esophageal perforations, mediastinitis, and at least 8 deaths to date have been attributed to these endoscopic techniques. Serious adverse events, including deaths, led to the American Gastroenterological Association Institute medical position statement recommending that "current data suggest that there are no definite indications for endoscopic therapy for GERD at this time"[87].

Surgical Management

Only surgical fundoplication can correct the physiologic factors contributing to GERD and prevent the need for long-term medication. Successful antireflux surgery involves (1) reducing the hiatal hernia back into the abdomen, (2) closing the opening in the diaphragmatic hiatus, (3) lengthening the intra-abdominal portion of the LES, and (4) strengthening the repair with a fundoplication. The most popular fundoplication is the 360° Nissen fundoplication. The partial posterior Toupet fundoplication is primarily used in patients who have aperistalsis or ineffective esophageal peristalsis. The latter is associated with less bloating and flatus, but not necessarily dysphagia when compared with a total fundoplication[89]. Most authorities in the United States believe the Nissen fundoplication is more durable.

Antireflux surgery has undergone a resurgence since the advent of the laparoscopic operation[90]. A systematic review identified six randomized controlled trials involving 449 patients that compared open and laparoscopic

fundoplication[91]. There was no significant difference in recurrence rates between the procedures, and laparoscopic fundoplication was associated with lower operative morbidity (NNT to prevent complication = 8; 95% CI, 3–16) and shorter hospital stay.

Indications for surgical fundoplication in the era of inexpensive PPIs and proven long-term safety of these drugs[89] are:

1. Patients who have typical or atypical GER symptoms who respond to PPIs but who want surgery because of
    - A desire for a permanent cure
    - Patient preference
    - An intolerance to PPIs
2. Failed medical therapy as a result of persistent volume regurgitation. Here heartburn symptoms are controlled, but regurgitation is a persistent problem.
3. Recurrent peptic strictures in younger patients
4. Respiratory complications related to regurgitation and recurrent aspiration

In patients who have Barrett esophagus, there is no convincing evidence that fundoplication reduces the long-term risk for esophageal adenocarcinoma[92].

Comparison studies of older medical treatments (antacids, H2RAs) consistently find surgical fundoplication better at healing esophagitis and relieving symptoms. There are few studies comparing fundoplication with

long-term PPI therapy, but one study[93] suggested that both were equally effective in controlling symptoms over 5 years, provided patients in the medical treatment group were allowed to increase the dose of the drug to twice daily if necessary.

Complications can occur after antireflux surgery, and many patients over time continue to require antireflux medications. In a database analysis, postoperative dysphagia was recorded in 19.4%, dilation was performed in 6.4%, and a repeat antireflux surgery was needed in 2.3% of patients. The surgical mortality rate was 0.8%. Approximately 50% of patients received multiple prescriptions for antireflux medications at a median of 5 years of follow-up evaluation after their surgery.

Tertiary specialized centers are seeing an increased rate of fundoplication failures[94]. The most common reasons for failure are herniation of the intact fundoplication into the chest, slipped fundoplication with a recurrent hiatal hernia, probably caused by a short esophagus, paraesophageal hernia through an intact fundoplication, too tight a fundoplication, and malpositioned fundoplication, usually on the cardia of the stomach. Total breakdown of the fundoplication is now rare. Revisional antireflux surgery needs to be performed by experienced surgeons, can be done laparoscopically but many prefer an open approach, and has increased morbidity and mortality compared with the initial operation.

AIMS OF THE STUDY

- To evaluate the association between Body Mass Index and Gastroesophageal Reflux Disease.

- To determine the correlation between Obesity and GERD in women.

REVIEW OF LITERATURE

OBESITY AND GERD

Obesity is increasing due to change in lifestyle and food habits among Indians. A recent survey of urban Asian Indians in Chennai (CURES) has estimated the prevalence of obesity as 45.9% using the cutoff value of BMI >23 as per the recent definition among a representative population of 2350 subjects[106].

WHO guidelines for Asians defines BMI >23 as overweight and >25 as obese[101]. However recommended action points for BMI in Asians have defined BMI >23 as overweight and >27.5 as obesity, with the cutoff for morbid obesity being >37.5[102].

A study which studied cutoff for BMI in Asians based on mortality risk has defined >23 as overweight and >25 as obese[103].

There are studies which advocate abdominal obesity rather than whole body measurement such as BMI as a marker of obesity since Indians are lean but tend to have increased abdominal fat

INTERHEART study has proposed waist hip ratio as a better marker of obesity than BMI[104].

There are other studies in favour of waist circumference as a better measure of obesity especially due to convenience of measuring it[105]

Studies indicate that gastro-esophageal reflux disease (GERD) is associated with obesity.

Effect of obesity on the pathogenesis of hiatus hernia which contributed to GERD was the focus of Wiad Lek et al in 1974[107].

Lower esophageal sphincter pressure (LESP) and esophageal function was studied in obese patients in 1980[108].

Effect of obesity on esophageal transit was published in 1985[110]. Gastroesophageal reflux and obesity association[109] and antireflux procedures such as the Angelchik antireflux prosthesis were studied in 1985[111].

Observation of the association between obesity and reflux disease, yielded conflicting conclusions among different studies; hence, the benefit of weight loss was also controversial. In a large, Swedish population interview-based study, Lagergren et al[112] concluded that the presence of gastroesophageal reflux symptoms occurred independently of BMI. However, this group (with the addition of Lindgren)[56] had previously linked the presence of gastroesophageal reflux as a risk factor for esophageal adenocarcinoma, and Lagergren et al[113] have shown that this complication of GERD is, in fact, strongly associated with BMI. In a similar population-based study, using a questionnaire among the residents of Olmstead County, Minnesota, BMI was independently associated with GERD[114]. Although earlier studies of the massively obese[115,116] and investigations of the effect of weight loss therapy in symptomatic improvement[117,118] were contradictory in their conclusions, further studies are in favour of the association.

Body mass index was strongly positively related to the frequency of symptoms of gastro-oesophageal reflux, occurring at least once a week in overweight participants compared with those of normal weight and obese people are almost three times as likely to experience these symptoms as those of normal weight according to the Bristol Helicobacter Project in UK which examined the relationship between body mass and GER symptoms[119].

Lifestyle factors, in particular overweight, obesity and smoking, were associated with increased reflux symptoms in a representative sample of the general adult population in a German survey[120].

A large study which compared ethnic groups found that obesity may disproportionately increase GERD-type symptoms in the white population and in male subjects rather than blacks and Asians[121].

The report of the Asia-Pacific consensus on the management of gastroesophageal reflux disease summarizes the conclusions and recommendations of a panel of gastroenterologists practising in the Asia-Pacific region. The group recognized that although gastroesophageal reflux disease (GERD) is less common and milder in endoscopic severity in Asia than in the West, there is nevertheless data to suggest an increasing frequency of the disease[122].

An update of the Asia-Pacific consensus in 2008 notes that GERD is increasing in frequency in Asia. Risk factors include older age, male sex, race, family history, higher socioeconomic status, increased body mass index, and smoking[123].

Non-erosive reflux disease (NERD) appears to be the most common form of GERD among Asian patients with differing predisposition to GERD among different ethnic groups[124].

The prevalence of erosive esophagitis and Barrett's esophagus in a multiethnic Malaysian population was studied, as well as the relationship of various factors associated with reflux disease. There was a preponderance of Indians with esophagitis; Indians had the highest prevalence of Barrett's esophagus[125].

Indian race and BMI were among the risk factors for GERD in an endoscopy based study in a multiracial Asian population[126].

Abdominal obesity was the significant risk factor for erosive esophagitis in a Korean endoscopy based study[127].

Another Japanese study showed similar conclusions that GERD and Hiatus hernia were related to obesity[128].

Asians tend to have a milder spectrum of the disease. Most Asian patients have non-erosive GORD; erosive oesophagitis is less commonly seen than in the Western population. Complicated GORD, such as oesophageal stricture and Barrett's oesophagus, is seldom encountered[129].

The ProGERD study found that higher BMI was associated with more frequent and more severe heartburn, regurgitation and esophagitis. Obese women, not men had esophagitis compared to women of normal weight[130].

The link between body mass and reflux is much stronger in women than in men, at least in the few studies that did sex-specific analyses. There is a significant association between body mass and symptoms of gastroesophageal reflux. The association is stronger among women, especially premenopausally, and use of hormone therapy strengthens the association, suggesting that estrogens may play an important role in the etiology of reflux disease[131].

A study which examined diet, lifestyle and gender found that females had significantly higher prevalence of GERD than males (66 vs. 48%). Obesity was significantly related to GERD[101].

Prevalence of overweightedness and obesity is increased in female but not in male patients with ascertained gastro-esophageal reflux disease in an Italian study which compared patients with general population[132].

Estrogen treatment alone, but not with progestin, may cause GER symptoms in postmenopausal women. Increasing weight and girth increases the risk of developing GER symptoms, whereas weight loss alleviates existing GER symptoms in post menopausal females on hormone replacement therapy[133].

Association between BMI and symptoms of gastroesophageal reflux disease in a large cohort of women and a dose–response relationship was observed for both frequent and infrequent symptoms, nocturnal symptoms, and

for all degrees of the severity and duration of symptoms that were studied. Moreover, weight gain was associated with an increased risk of symptoms of gastroesophageal reflux disease, and weight loss was associated with a decrease in risk[134].

Esophagitis was significantly more prevalent in obesity than in normal subjects and association between GER and obesity remained significant adjusting for medication use in an endoscopy based study[135].

Oesophageal transit in obese reflux patients is slower than in their leaner counterparts,[136] BMI is associated with the development of a hiatal hernia[137,138] (an important factor in delaying the clearance of acid from the oesophagus) and there is evidence that increasing BMI increases intra-abdominal pressure,[139,140] although this may be counteracted by equivalent increases in lower oesophageal sphincter pressure.

Exposure of the distal esophagus to pathologic levels of refluxed gastric juices causes the clinical phenomenon of gastroesophageal reflux disease (GERD) and its sequelae. Reflux is prevented by a mechanically competent lower esophageal sphincter (LES), which provides a barrier between the gastric and esophageal compartments, and a proper clearance activity of the esophageal body by appropriate LES relaxation and peristaltic contractions on swallowing[141,142].

Despite the presence of a structurally normal LES and effective esophageal clearance, excessive gastroesophageal reflux and consequent esophageal damage may still occur. In these patients, other factors must therefore exist that override the standard barrier to reflux.

GERD may be caused by an external alteration in the anatomical and physiological characteristics of the LES or esophageal body. Excessive fat deposition could interfere directly with the LES esophageal body complex, preventing it from functioning effectively, or it could change the intra-abdominal pressure dynamics and render the barrier incompetent.

The presence of excess fat in and around the gastroesophageal junction could alter the anatomical structure and, hence, the geometry of the cardia, placing the sphincter at a mechanical disadvantage to counter gas distension forces attempting to pull it open. The acute angle of His, an important anatomical structure preventing gastric wall tension from pulling the LES apart[143] may become blunted, thus enabling moderate levels of gastric distension to more readily induce transient sphincter opening. Similarly, the potentiation effect on sphincter competency derived from the diaphragmatic crural sling[144] may be attenuated if this region is surrounded by cushions of fatty tissue.

Obese individuals have a higher intra-abdominal resting pressure, and this relates to the sagittal abdominal diameter[145]. Excess fat deposition in and around abdominal viscera, in addition to elevating intra-abdominal pressure, may interfere with and delay gastric emptying[146]. This promotes fundic

distension, with corresponding sphincter unfolding and length shortening, reducing its ability to function as an effective barrier[147]. The restriction of free space within the peritoneal cavity is likely to result in reduced pressure compliance within the abdominal compartment. Fluctuations in the intra-abdominal pressure, such as occurs with positional or postural change, coughing, or straining, are therefore exaggerated, with sudden sharp rises in gastric pressure overcoming LES resistance.

Retrospective analysis of body mass index in patients with normal esophageal manometric findings but with symptomatic and objectively confirmed gastroesophageal reflux found a strong correlation between body mass index and severity of gastroesophageal reflux. Patients who were overweight had significantly higher distal esophageal acid exposure[148].

A study to analyze the relationship between obesity and the morphology of the esophagogastric junction (EGJ) pressure segment using high-resolution manometry: The association between anthropometric variables and pressure values were examined there was a significant correlation of body mass index (BMI) and waist circumference (WC) with intragastric pressure and gastroesophageal pressure gradient (GEPG) Multivariate analysis adjusting for age, gender, and patient type did not alter the direction or magnitude of this relationship. In addition, obesity was associated with separation of the EGJ pressure components.Obese subjects are more likely to have EGJ disruption (leading to hiatal hernia) and an augmented GEPG providing a perfect scenario for reflux to occur[149]

During the postprandial period, both obese and overweight patients had substantial increase in 2-hour rate of transient lower esophageal sphincter relaxation (TLOSR) with acid reflux and gastroesophageal pressure gradient (GOPG). BMI and waist circumference significantly correlated with TLOSR. Obesity is associated with increased TLOSR and acid reflux during the postprandial period. Abnormal postprandial LOS function may be an early event in the pathogenesis of obesity-related GERD[150].

Obesity may promote the development of GERD through the mechanical stresses imposed on the antireflux barrier, specifically increased pressure gradients across the esophagogastric junction (EGJ) and the propensity to develop hiatus hernia. However the causation is multifactorial and a single anatomical abnormality cannot account for GERD[151].

Increase in BMI has been shown to be associated with increase in prevalence of GERD symptoms, esophageal mucosal injury, and GERD complications by increased intra gastric pressure, GE pressure gradient, esophageal motor and sensory abnormalities, increase in prevalence of hiatus hernia, increase in serum female hormones, diet and increase in comorbidities. It is highly likely that multiple factors contribute to the prevalence of GERD in the obese patient[152].

Abdominal obesity rather than BMI is an independent risk factor for erosive esophagitis in the Korean population[153].

Obesity, especially abdominal obesity, was the significant risk factor for erosive esophagitis[154]. When 3363 patients who had GERD related esophagitis were analysed in a Korean study.

Waist circumference, but not BMI, had some modest independent associations with the risk of Barrett's esophagus. The findings provide partial support for the hypothesis that abdominal obesity contributes to GERD, which may in turn increase the risk of Barrett's esophagus[155].

Weight loss, through caloric restriction and behavioral modification, has been studied infrequently as a means of improving reflux. Bariatric surgery and its effects on a number of obesity-related disorders have been studied more extensively. Roux-en-Y gastric bypass (RYGB) has been consistently associated with improvement in the symptoms and findings of GERD. The mechanism of action through which this surgery is successful at improving GERD may be independent of weight loss and needs further examination. Current evidence suggests that laparoscopic adjusted gastric banding should be avoided in these patients, as the impact on gastroesophageal reflux disease appears unfavorable[156].

MATERIALS AND METHODS

A total of 106 consecutive patients referred to the Gastroenterology unit with symptoms of GERD were included in this study. The study period was from July 2007 to July 2008.

Ethical committee approval and consent from the patients were obtained.

STUDY DESIGN

Prospective analysis of BMI, waist-hip ratio in patients with symptomatic GERD.

SELECTION CRITERIA

Consecutive patients attending gastroenterology outpatient department at Stanley Government Medical College Hospital for symptoms of GERD were included in the study.

Patients who had dysmotility, those with history of abdominal surgery and pregnant women were excluded from this study.

DEMOGRAPHIC DATA

Total of 106 patients of which 45 were males and 61 females.

Age ranged from 21 to 78 years. Mean age was 47.7

Age and gender distribution of the study subjects are shown in Charts 1 & 2.

Symptoms Defining Gastroesophageal Reflux Disease

Classic symptoms of GERD such as heartburn and/or regurgitation,and other symptoms such as dysphagia, noncardiac chest pain, water brash, odynophagia, burping, bloating, early satiety, hiccups, nausea, vomiting, asthma, hoarseness of voice, chronic cough, recurrent lower respiratory symptoms, caries teeth, weight loss, upper GI bleed, and history of other comorbid illnesses especially related to obesity were recorded after direct questioning.

History regarding diet habits such as intake of fatty food, fried food, spicy food, quantity of meals, excessive intake of citrus fruits, beverages such as coffee and tea, chocolates, aerated soft drinks, smoking, alcohol consumption, tobacco chewing, use of NSAIDs, oral contraceptives were recorded.

History regarding radiation treatment, prolonged naso gastric aspiration and previous peptic ulcer disease were also recorded.

Anthropometric Indices Were Recorded

Weight was measured using a standard spring balance type of weighing machine; height measured using a stadiometer on even ground; waist,hip circumference and abdominal girth were measured using a flexible nonelastic

type of measuring tape. Abdominal girth was measured at the level of umbilicus in supine posture. Waist circumference was measured as the narrowest part of the torso in standing position and hip circumference as the widest part at the level of buttocks. Waist hip ratio was calculated. BMI was calculated : weight(kg)/height(m2)

Patients were then assigned to four categories according to their BMI.

Statistical analysis was done for quantitative variables and expressed as mean +/- SD.

The significance of differences in gender according to BMI, waist circumference and waist hip ratio were assessed using chi squared test. P value less than 0.05 was considered statistically significant.

RESULTS

Of the 106 patients presenting with GERD.

52(49.1%) had BMI <23, out of which 14 were underweight and 38 were of normal BMI.

54(50.9%) had BMI >23, among which 22 were overweight and 32 were obese.

TABLE-1 : Table showing BMI distribution

BMI	<18.4	18.5 - 22.9	23 - 24.9	>25
No.	14	38	22	32
%	13.2	35.85	20.7	30.2

If the data on gender were considered, females outnumbered male overweight and obese GERD patients.

There were 40(65.6%) females vs 14(31.1%) males among GERD cases with BMI >23.

Odds ratio for obesity in females was 0.2 with 95% CI 0.1-0.5.

Risk ratio was 0.5 with 95% CI 0.3-0.8

Of the 45 males with GERD, 16(35.6%) had waist circumference >87cm, whereas 39(63.9%) out of 61 females had waist circumference >82.

When waist hip ratio was calculated, 20(44.4%) out of 45 male GERD patients were above 0.9 and 51(83.6%) out of 61 female GERD patients were above 0.8.

The following table shows the gender based data for those who exceeded normal values of BMI, waist circumference and waist-hip ratio.

TABLE-2 : Table showing comparison between male and female patients exceeding reference range for BMI, waist circumference and waist- hip ratio

Gender	Total	BMI >23 % (No.)	WC >87 & 82 cm % (No.)	W-H R >0.9 & 0.8 % (No.)
Male	45	31.1% (14)	35.6% (16)	44.4% (20)
Female	61	65.6 % (40)	63.9% (39)	83.6% (51)
Pvalue		0.01	0.06	0.02

The number of male GERD patients who were of <23 BMI(31) Were significantly more than those with BMI in overweight or obese category (14): P value 0.008.

However the number of female GERD patients who were overweight or obese (40) exceeded those with BMI <23(14) P value 0.02.

The number of male GERD patients with waist circumference <87cms (29) were significantly more than those with higher waist circumference (16): P value 0.03, whereas the number of females with waist circumference >82(39) were more than those within normal range(22) but this value was not statistically significant.

The number of females(39) outnumbered the number of Males (16) in the high waist circumference group but the difference was not statistically significant; the number of males (29) were more than females(22) in the normal waist circumference group: P value 0.05.

The number of male GERD patients who were within reference range for waist-hip ratio (25) were significantly more than those with higher waist-hip ratios (20): P value 0.001. However the number of female GERD patients with higher waist-hip ratios(51) exceeded those within reference range(10): P value 0.006.

The number of females(51) were more than males(20) in the higher waist-hip ratio range: P value 0.02 whereas the number of males (25) were more in the normal waist-hip ratio levels compared to females(10): P value 0.01

TABLE-3 : Table showing distribution of male GERD patients based on BMI, waist circumference (WC) and waist-hip ratio(W-H R)

BMI	**<23**	**>23**
No.	**31**	**14**
WC	**<87cm**	**>87cm**
No.	**29**	**16**
W-H R	**<0.9**	**>0.9**
No.	**25**	**20**

TABLE-4 : Table showing distribution of female GERD patients based on BMI, waist circumference (WC) and waist-hip ratio (W-H R)

BMI	<23	>23
No.	21	40
WC	<82cm	>82cm
No.	22	39
W-H R	<0.8	>0.8
No.	10	51

Upper GI endoscopy was done in 99 patients out of which there were 42 males and 57 females.

Patients were classified as having erosive(ERD) vs nonerosive(NERD) reflux disease.

There were more males with NERD than ERD; p value:0.03.

There were more females with NERD than ERD; p value:0.07.

Males were more likely to have ERD compared to females.

OR:4.7 95% CI 1.6-13.6;pvalue:0.003.

7 out of the 15 males with ERD were smokers.

TABLE-5 : Table showing comparison between male and female patients with NERD vs ERD

Variables	NERD	ERD
Total No.	78	21
Male	27	15
Female	51	6
BMI>23	44	10
Male	8	6
Female	36	4

56.4% of NERD patients were of BMI>23; 46.6% of ERD patients were of BMI>23.

Out of 78 patients with NERD 70.6% of females and 29.6% of males had BMI >23.

Out of 21 patients with ERD 66.6% of females and 40.0% of males had BMI >23.

Hiatus hernia was present in 13 patients out of which 7 were males and 6 were females. 4 in each group had BMI >23

TABLE-6 : Table showing distribution of Hiatus hernia

Variables	No.	% (no.) BMI>23	% (no.) WC>87/82	% (no.) W-H R>0.9/0.8
Total	**13**	**61.5% (8)**	**92.3% (12)**	**84.6% (11)**
Male	**7**	**57.1% (4)**	**85.7% (6)**	**71.4% (5)**
Female	**6**	**66.7% (4)**	**100% (6)**	**100% (6)**

Lax LES was present in 6 males and 5 females, out of which 1 male and 4 females had BMI >23 .

TABLE-7 : Table showing distribution of Lax LES

Variables	No.	% (no.) BMI>23	% (no.) WC>87/82	% (no.) W-H R>0.9/0.8
Total	**11**	**45.4% (5)**	**45.4% (5)**	**63.6% (7)**
Male	**6**	**16.7% (1)**	**16.7% (1)**	**50% (3)**
Female	**5**	**80% (4)**	**80% (4)**	**80% (4)**

Chart – 1

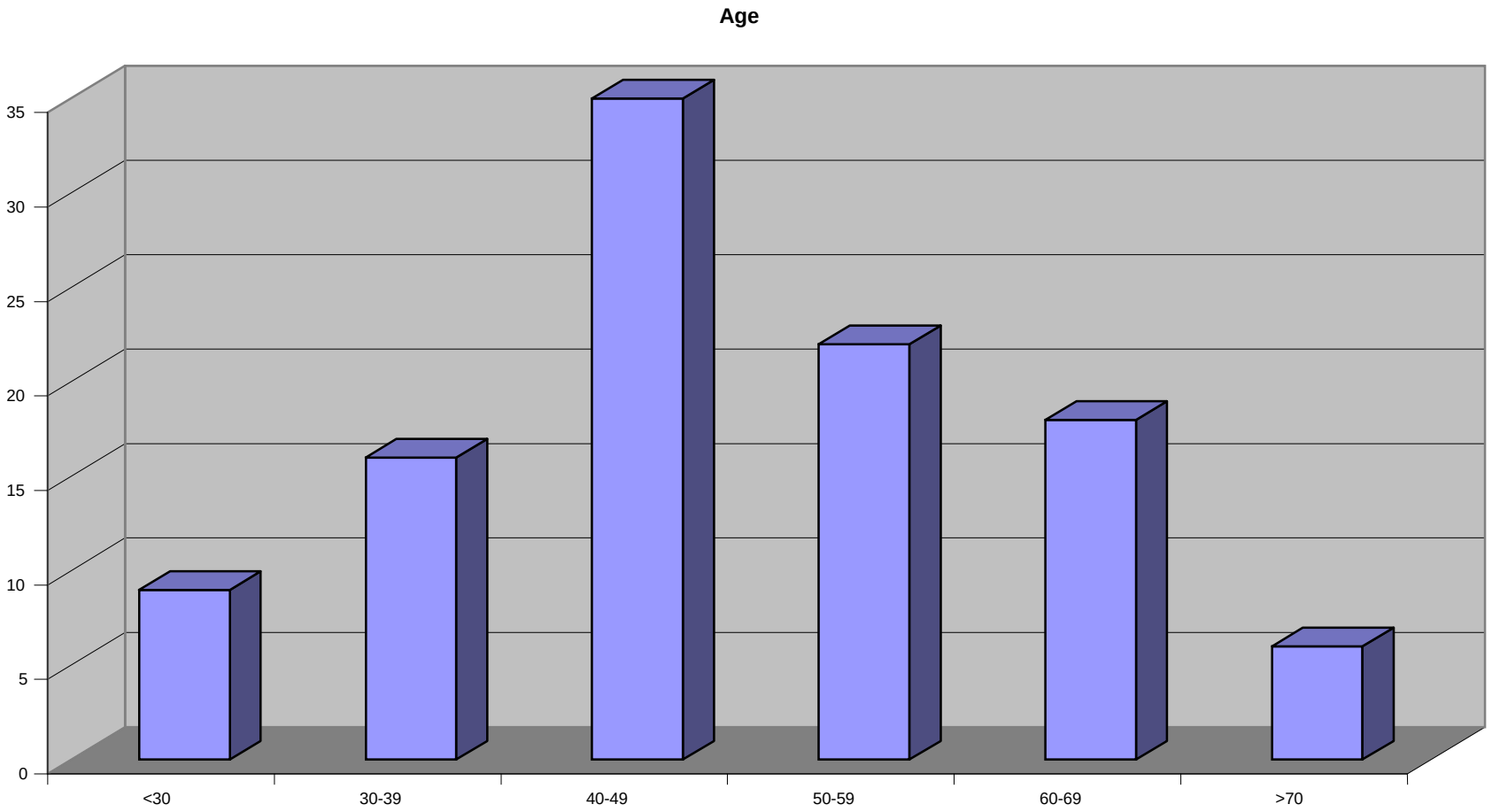


Chart – 2

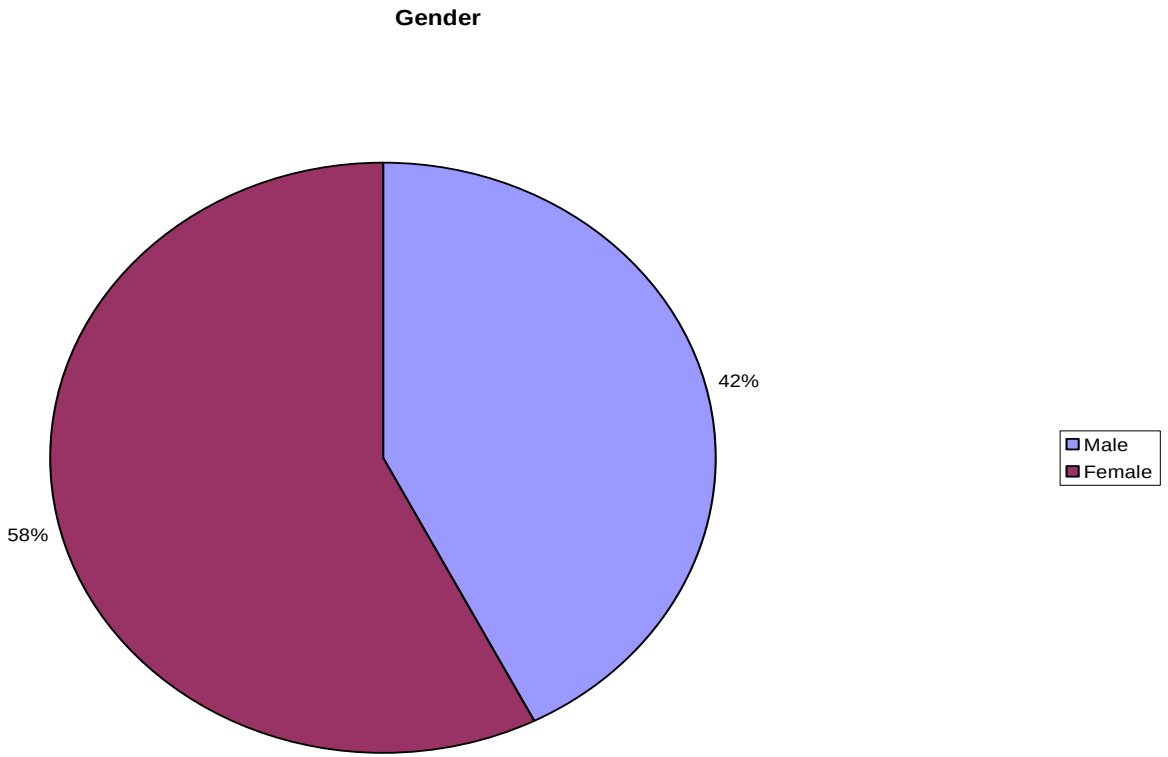


Chart – 3

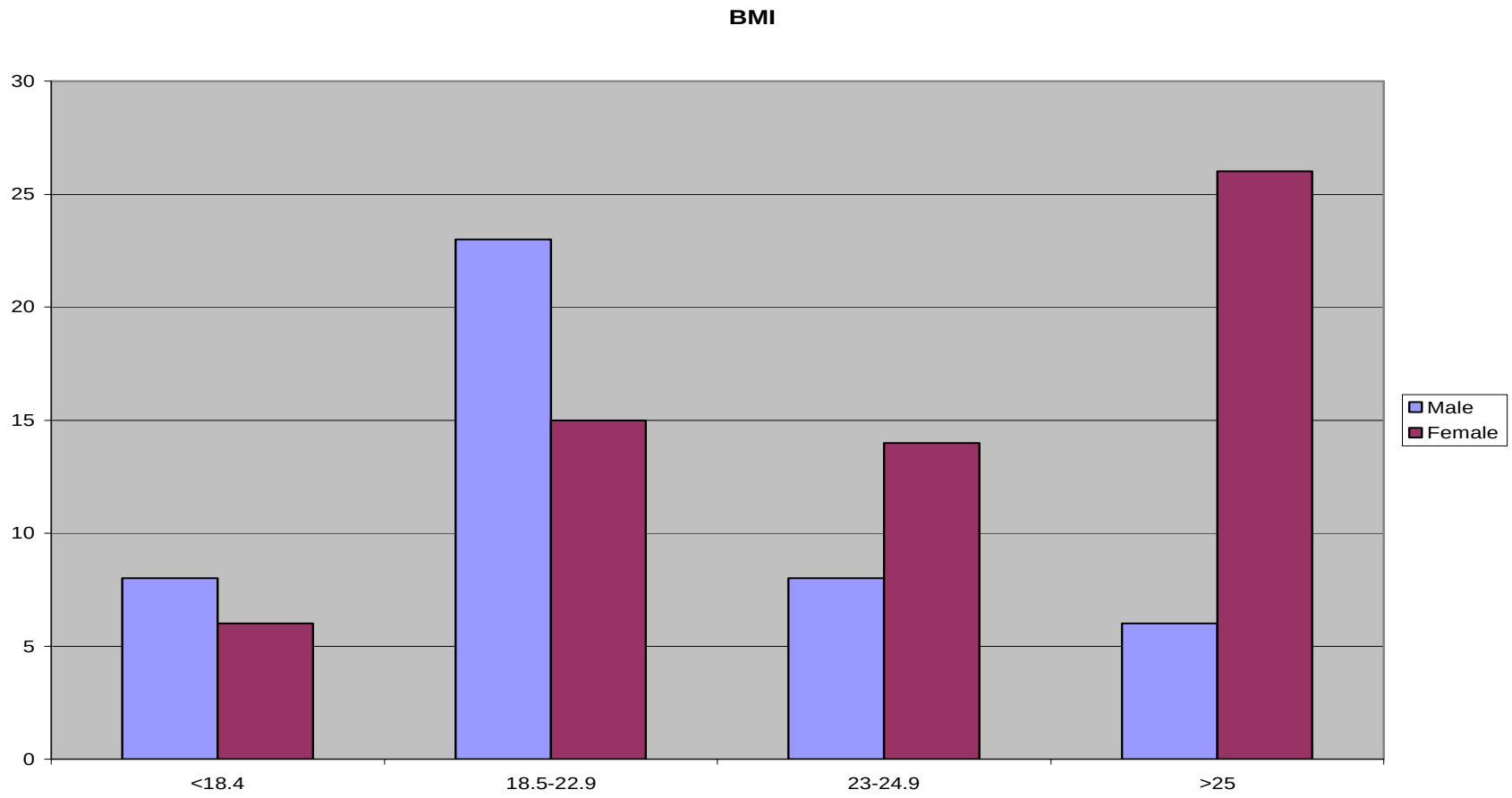


Chart – 4

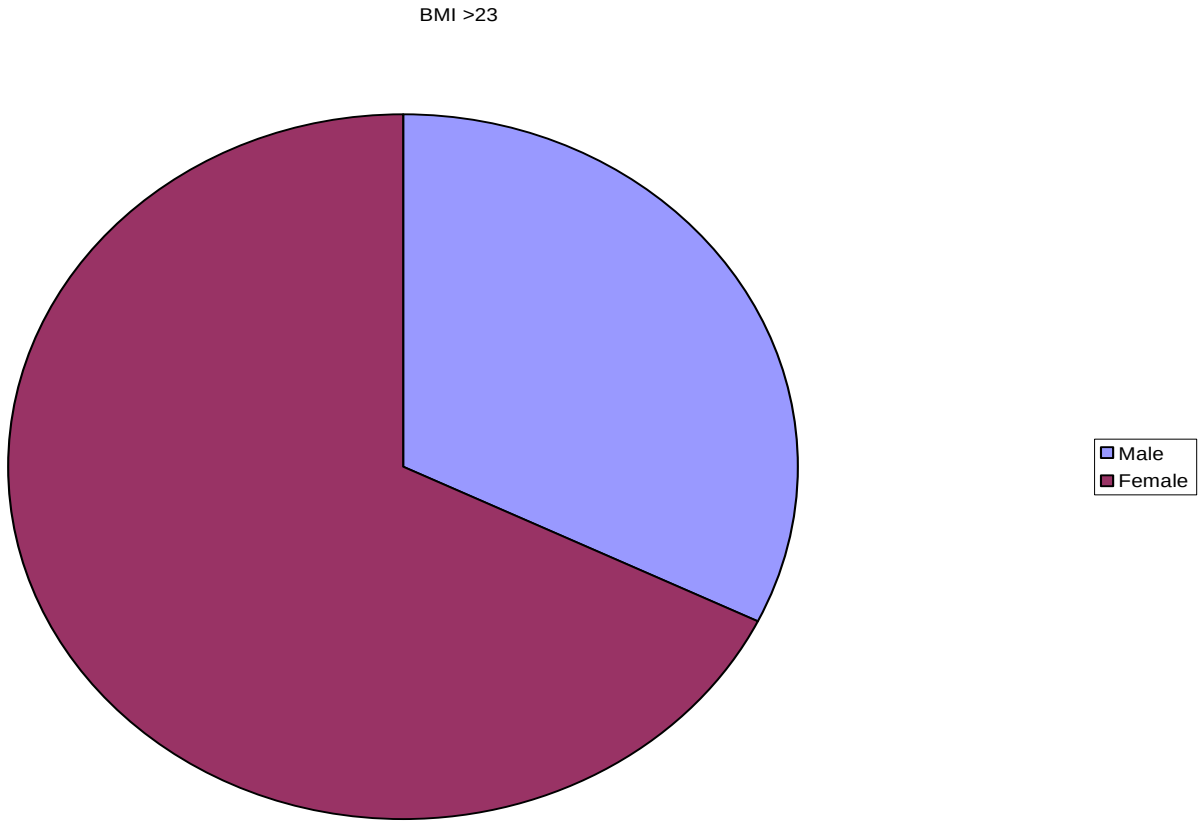
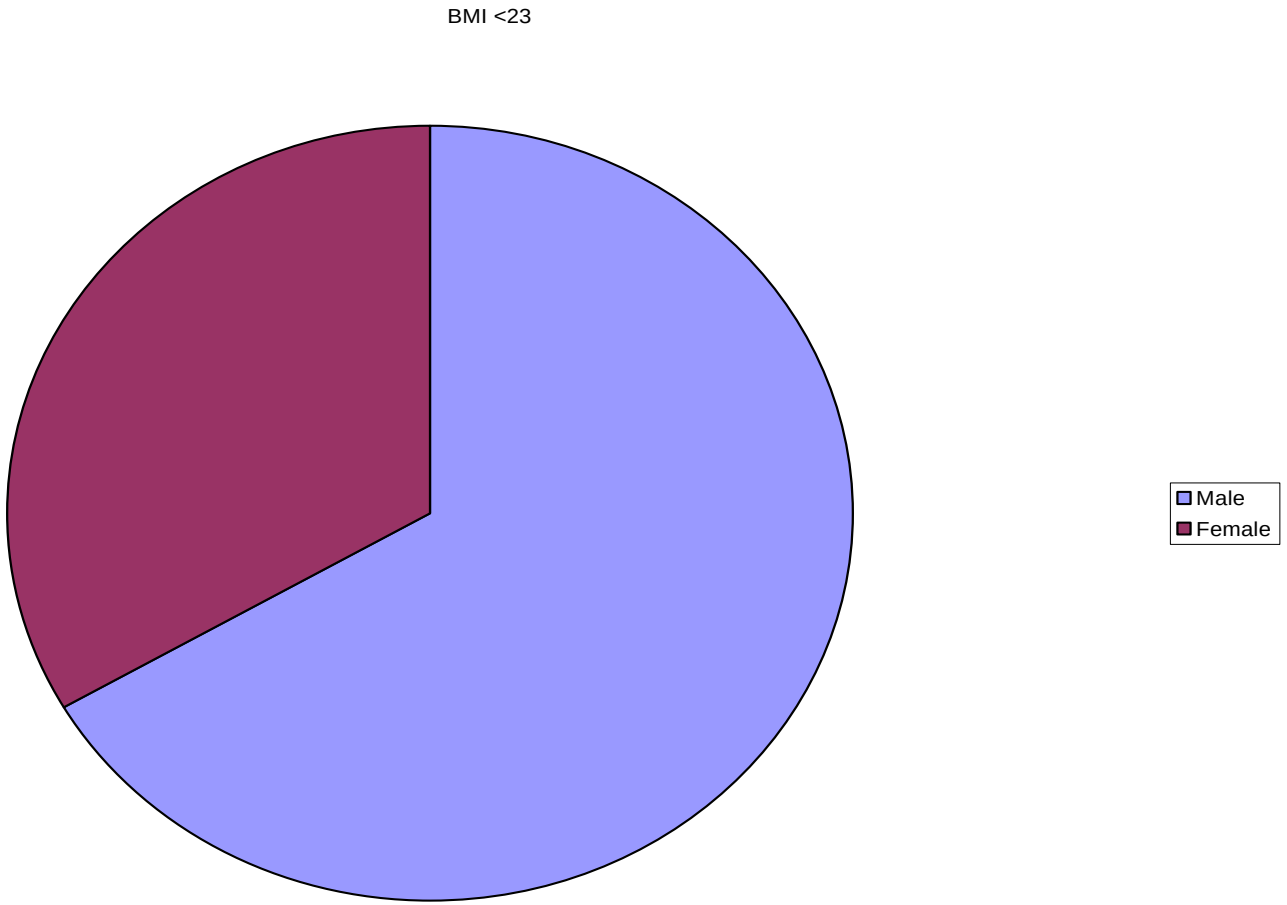


Chart – 5



SUMMARY

- A total of 106 GERD patients were analysed for prevalence of obesity by measures such as BMI, waist circumference and waist-hip ratio.
- There were 45 males and 61 females.
- BMI was >23 in 54 of 106 patients:50.9%
- There were significantly more females with high BMI and waist-hip ratio compared to males; however the gender difference was not statistically significant based on waist circumference.
- The number of males with BMI, waist circumference and waist-hip ratio less than cutoff value for obesity were significantly more than obese males.
- The number of females with BMI and waist-hip ratio more than cutoff value for obesity were significantly more than nonobese females; however, the difference between obese and nonobese female patients based on waist circumference was not statistically significant.
- 99 patients underwent UGI endoscopy.
- NERD was more prevalent than ERD in both males and females.
- Males were more likely to have ERD compared to females.

DISCUSSION

Association Between Obesity And Gerd

Gastro esophageal reflux disease is a multifactorial process resulting from an imbalance between defensive factors (antireflux barriers, esophageal acid clearance,tissue resistance) and aggravating factors (gastric acidity,volume and duodenal contents). Obesity satisfies several criteria for a causal association with GERD and abdominal obesity impairs antireflux function by increasing intragastric pressure, gastroesophageal gradient,TLOSR and esophageal acid exposure.

El-Serag[157] reviewed epidemiological data to find a consistent association of obesity with GERD symptoms, erosive esophagitis and esophageal adenocarcinoma.

The Asia Pacific consensus update states that GERD is increasing in frequency in Asia. Risk factors include increased body mass index. Weight loss improves reflux symptoms[123]

Factors Defining Obesity

BMI has been widely used as an indicator of adiposity; its limitations have been widely recognized by its dependence on race: Asians have larger percentage of body fat at lower BMI values.As compared to BMI, waist circumference and waist-hip ratio have been used as surrogates of body fat centralization.Many studies such as the INTERHEART have proposed the use of waist circumference and waist-hip ratio as markers of obesity rather than BMI.

In this study, obesity was defined according to the WHO cutoff as BMI >25 and overweight as BMI >23 irrespective of gender[102]

Waist circumference and waist-hip ratio cut points were defined as >87 in males, >82 in females and >/=0.9 in males and >/=0.8 in females respectively as per current recommendations for urban Asian Indians[158]

Age Distribution

The age range of 106 consecutive patients with GERD was 21-78, mean age was 47.7 Age distribution is shown in Chart-1

Gender Distribution

There were 58% females and 42% males. Dore MP et al report a similar gender distribution with 66% females vs 48% males in their cohort[148]. Female preponderance has been noted in various studies possibly due to the effect of Estrogen[131,132,133,159]. Gender distribution is shown in Chart-2.

BMI Distribution

The BMI values in this group ranged from 15.2-35.4 with mean BMI 22.7 and median BMI 23.0 as compared to the BMI range of 16-52 in a western cohort with mean BMI 27.5 and median BMI 27 in a study by El-Serag et al in USA[159]. The lower mean BMI in Asian population argues against the use of same BMI cutoff values for different ethnic populations[101,102,103,104,105,106,158]. The patients were classified according to their BMI as thin (<18.5) who were 13.2%, normal(18.5-23) with 35.85%, overweight(23-25) with 20.75% and obese(>25) with 30.2% of the patients. The BMI distribution in this study is represented in Chart-3.

The BMI distribution of patients in a study by Sakaguchi et al[128] in Japan showed 20.96% in the thin group which is marginally more than this study. The patients in normal BMI group was 24.42% which was less compared to this study. The patients with BMI >23 who were overweight and obese were 31.86% in the Japanese study whereas those with BMI >23 were 50.95% in this study.

A Korean study which classified GERD patients as per western standards found the distribution to be BMI <25 (68.9%), 25-30(28.7%) and >30(2.4%)[153].

Corley DA et al found an odds ratio of 1.52 for overweight and 2.15 for obese group for risk of GERD in a meta-analysis of studies within the USA[11]. whereas a study in Asians in Korea found an odds ratio of 1.2 for overweight and 1.9 for obese patients with GERD[160].

The age-wise mean BMI was analysed and the mean BMI was highest in the 50-59 age group (25.41) of patients while the most number of patients with GERD was found in the 40-49 age group (n35) in this study. In Locke et al study in USA[114] the mean age was 50 and <24 BMI group were 15%, overweight were 37% and obese were 30% similar to this study with obese 30.2%.

Gender Differences in BMI Distribution

Female GERD patients in all age groups had higher mean BMI compared to males, the highest mean BMI(28.24) was observed in 50-59 age group.

The number of female overweight and obese GERD patients were more than males in the same BMI group: 40(65.6%) vs 14(31.1%).The difference was statistically significant.P value 0.01 This is supported by a study in Italy by Piretta L et al who found increased prevalence of overweightedness and obesity in females with GERD[132]

The gender difference based on BMI is shown in Charts 4 & 5.

The number of obese females having GERD based on BMI was significantly more than nonobese females.40(65.6%) vs 21(34.4%). P value 0.02.

The converse was true for males,with 14(31.1%) in obese group vs 31(68.9%) in nonobese group. P value 0.008.

Waist circumference and waist-hip ratio as risk factors for GERD:

The prevalence of obesity was more based on waist circumference (51.9%) and waist-hip ratio (67%) than BMI (30.2%) in this study

Many studies have shown a positive correlation for waist circumference with reflux disease. El-Serag et al have correlated waist girth with increased intragastric pH.[159]

Corley DA et al found correlation of symptoms with abdominal girth in among whites but not blacks or Asians in USA[121] and symptom severity increased with abdominal girth[155] and the odds ratio was 1.86,

When gender specific data were considered, females with higher waist circumference were more than males: 39(63.9%) vs 16(35.6%) P value 0.06 but the difference was not statistically significant.

Females with waist-hip ratio above cutoff levels were more than males: 51(83.6%) vs 20(44.4%) with significant difference: P value 0.02

The number of obese females based on waist-hip ratio was significantly more than nonobese females in this study: 51(83.6%) vs 10(16.4%) P value 0.006.

Endoscopy Findings

The 99 patients who underwent endoscopy were classified as erosive(ERD) and non-erosive reflux disease(NERD).

In this study 78 patients(78.8%) had NERD and 21(21.2%) had ERD. 15(35.7%) out of 21 patients with ERD were males with odds ratio 4.7 (95% CI: 1.6-13.6) P value 0.005.

NERD was more prevalent than ERD in both males and females.

This was comparable to an Asian endoscopy based study by Rosaida MS et al who have shown a similar incidence of NERD 65.5% and ERD 13.4%[126]

Male Gender As a Risk For ERD

Rosaida MS et al[126] have found male gender being a risk factor for ERD. Moki F et al also found males to be at risk for ERD with odds ratio 2.5 and obesity to be associated with ERD; odds ratio 1.9[161]

Nocon et al found obese women to be at risk for ERD compared to nonobese women with odds ratio 2.5 but in the present study this difference was not statistically significant.[130]

Obesity as A Risk For ERD

Zafar et al found endoscopic severity to correlate with BMI in 203 subjects in a study in Pakistan[162]

Wilson et al found BMI to correlate with ERD: odds ratio 1.8[138].

Some studies have found waist hip ratio to be a risk factor for ERD[154] with odds ratio 2.3.

In a Korean study Kang MS et al found abdominal obesity rather than BMI to correlate with risk for ERD: odds ratio 2.3 The prevalence of abdominal obesity in this study was 24.2% and ERD was 6.6%[153]

In the present study there was no significant correlation between obesity and ERD with BMI, waist circumference or waist-hip ratio as criteria for obesity in either gender although the prevalence of ERD was 21.2% of the 99 patients who underwent UGI endoscopy.

Hiatus Hernia and GERD

Hiatus hernia was present in 13(13.1%) patients.Rosaida MS et al have reported a prevalence of 6.7% in their group in Malaysia[126]

Wilson LJ et al have reported an association between hiatus hernia and risk of ERD with odds ratio 4.2[138]

In the present study 5 patients had hiatus hernia among the 21 with ERD. The odds ratio was 2.7 but the association was not statistically significant.

Table showing comparison of previous studies with present study

Variables	Previous studies	Present study
AGE		
Range		21-78
Mean	50 (Locke[114])	47.7
GENDER		
Females	66% (Dore[148])	58%
Males	48% ’’	42%
BMI		
Range	16-42 (El-Serag[159]	15.2-35.4
Mean	27.5 ”	23.0
BMI Distribution		
Thin <18.5	20.9% (Sakaguchi[128])	13.2%
Normal 18.5-23	24.42% ”	35.85%
Overweight 23-25	} 31.86% ”	20.75%
Obese >25		30.2%
OBESITY	30% (Locke[114])	30.2%
NERD	65.5% (Rosaida[126])	78.8%
ERD	13.4% ”	21.2%
MALE GENDER as risk for ERD	OR 2.5 (Moki[161])	4.7
HIATUS HERNIA	6.7% (Rosaida[126])	13.1%
HIATUS HERNIA as risk for ERD	OR 4.2 (Wilson[138])	2.7

CONCLUSIONS

- ❖ This study adds to a growing body of literature that strongly suggests an association between obesity and gastroesophageal reflux disease.
- ❖ Prevalence of obesity among patients with GERD was more based on waist circumference and waist-hip ratio than BMI.
- ❖ The link between obesity and GERD is stronger in women.

Implications of this study is that:

a) Clinicians should ask about symptoms of GERD when assessing the health risks in overweight and obese patients.

b) Weight reduction should be advised in those with reflux symptoms as Barrett's esophagus and esophageal cancer are known risks of long standing reflux.

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ABBREVIATIONS

BMI	body mass index
Oe N/I/E/U	oesophagus normal/ inflammation/erosion/ulcer
St N/I/E/U	stomach normal/inflammation/erosion/ulcer
Du N/I/E/U	duodenum normal/inflammation/erosion/ulcer
M	male
F	female
abd girth	abdominal girth
W-H ratio	waist-hip ratio
NV/week	nonvegetarian diet:no. of times per week
NSAID	nonsteroidal anti-inflammatory drugs
NG aspir	nasogastric aspiration
D/N	day/night
D/Occ	daily/occasionally
D/W/M	daily/weekly/monthly
int/cont	intermittent/continuous
sol/liq	solids/liquids
rec LRI	recurrent lower respiratory infections
UGI	upper gastrointestinal

MGE No.	medical gastroenterology serial number
L S mod	life style modification
Resp to Rx	response to therapy
rec	recurrence of symptoms on stopping treatment
lax LES	laxity of lower esophageal sphincter
DM	diabetes mellitus
Htn	hypertension
Hypothyr	hypothyroidism

P R O F O R M A

1. Name :
2. Age :
3. Gender : Male / Female
4. Occupation :
5. Address :
6. M.G.E No. :
7. Height : Cms
8. Weight : Kgs
9. BMI :
10. Abdominal girth :
11. Waist-hip ratio :
12. Smoking : Yes / No No. Years
13. Tobacco chewing : Yes / No
14. Alcohol use : Yes / No Frequency Duration
15. Diet : Veg. / Non.Veg Non.Veg. No.of times/week
16. Fried food : Yes / No No. of times/week
17. Type of Food : Bland/Mildly spicy/moderately spicy/very spicy
18. Quality of Food : Small / Moderate / Large
19. Citrus fruits : Yes / No
20. Fatty Food : Yes / No
21. Chocolates : Yes / No
22. Coffee / Tea : Yes / No No.of times/day
23. Aerated/Soft drinks : Yes / No No.of times/day
24. NSAID use : Yes / No
25. Pill : Yes / No
26. Infection : Yes / No
27. Radiation : Yes / No

28. Pregnancy : Yes / No

29. Prolonged NG aspiration : Yes / No

30. Peptic Ulcer / ZES : Yes / No

31. Scleroderma : Yes / No

32. Heartburn : Duration

Day / Night / both

Everyday / No.of times/day: /week: /Month

Supine / bending over / sitting

33. Regurgitation : Yes / No

Duration

Day / Night / both

Everyday / No.of times/day: /week: /Month

Supine / bending over / sitting

Post meal regurgitation: Yes / No

34. Dysphagia : Yes / No

Intermittent / continuous

Solids / Liquids / Both

35. Chest pain : Yes / No

Duration

Frequency

Severity

Related to meal : Yes / No

Stress: Yes / No

Sleep: Yes / No

Relief: Spontaneous/Anatacids/PPI/H2RA

36. Water brash : Yes / No

37. Odynophagia : Yes / No

38. Burping : Yes / No

39. Bloating : Yes / No

40. Early satiety : Yes / No

41. Hiccups : Yes / No

42. Nausea : Yes / No

43. Vomiting : Yes / No

44. Asthma : Yes / No

45. Hoarseness of voice : Yes / No

46. Chronic cough : Yes / No

47. Recurrent LRI : Yes / No

48. Caries teeth : Yes / No

49. Weight loss : Yes / No

50. UGI Bleed : Yes / No

INVESTIGATIONS:

51. Endoscopy No. :

Result : Normal / Edema / Erythema / Friability / Red streaks / erosions / Ulcers

52. Barium study :

53. Therapy :

53.1 Change in diet : Yes / No

53.2 Relief with raising head-end of bed : Yes / No

53.3 Antacids : Yes / No

53.4 H2RA : Yes / No

53.5 PPI : Yes / No

54. Recurrence of symptoms on stopping drugs: Yes / No

MASTER CHART

Sl.No	MGE No	Age	Gender	Occupation	Place	Height	Weight	BMI	abd girth	waist/hip	W-H ratio	Smoking	Tobacco	Alcohol	NV/Week	friedfood/w	quality	quantity
1	2249/02	62	F	housewife	Chennai	155	56	23.31	81	88/97	0.91	No	Yes	No	3	1	spicy	moderate
2	5792/02	67	F	housewife	Chennai	148	51	23.28	84	85/100	0.85	No	No	No	1	1	spicy	moderate
3	6251/02	34	M	driver	Chennai	158	50	20.03	77	78/87	0.89	No	No	No	1	1	spicy	moderate
4	6357/02	49	M	security	Chennai	178	51	16.09	67	71/85	0.80	Yes	No	Yes	2	2	spicy	moderate
5	1334/03	71	F	housewife	Chennai	147	41	18.97	78	72/90	0.80	No	No	No	1	>3	spicy	moderate
6	2779/03	46	M	driver	Chennai	162	40	15.24	64	62/74	0.83	Yes	No	Yes	>3	>3	spicy	large
7	3605/03	78	M	-	Chennai	164	45	16.73	71	73/84	0.86	Yes	No	Yes	2	>3	spicy	large
8	4858/03	68	F	housewife	Chennai	142	53	26.28	87	88/100	0.88	No	No	No	2	2	spicy	moderate
9	302/03	46	F	housewife	Chennai	150	68	30.22	97	98/111	0.88	No	No	No	1	<3	spicy	moderate
10	2806/04	62	F	maid	Chennai	149	44	19.82	74	84/92	0.91	No	No	No	3	>3	spicy	moderate
11	3780/04	62	F	maid	Chennai	144	54	26.04	94	94/102	0.92	No	Yes	No	3	<3	spicy	large
12	4005/04	35	F	housewife	Chennai	152	61	26.40	82	89/98	0.90	No	No	No	0	0	spicy	large
13	4095/04	41	M	-	Chennai	177	58	18.51	74	81/91	0.89	No	Yes	No	2	0	spicy	moderate
14	4729/04	50	M	clerk	Chennai	162	66	25.15	91	87/97	0.89	Yes	No	No	3	>3	spicy	moderate
15	5995/04	74	F	housewife	Chennai	144	44	21.22	74	78/84	0.93	No	Yes	No	0	0	spicy	moderate
16	754/05	45	F	housewife	Chennai	161	60	23.15	78	82/95	0.86	No	Yes	No	1	2	spicy	large
17	1068/05	52	F	housewife	Chennai	146	75	35.18	93	96/112	0.86	No	No	No	1	<3	spicy	moderate
18	1106/05	55	M	security	Chennai	165	65	23.87	96	93/97	0.96	No	Yes	Yes	0	0	spicy	moderate
19	2599/05	60	F	housewife	Chennai	154	46	19.39	86	89/91	0.98	No	No	No	2	<3	spicy	large
20	2754/05	45	F	housewife	Chennai	151	69	30.26	94	93/106	0.88	No	No	No	3	<3	spicy	large
21	2936/05	45	F	labourer	Chennai	153	48	20.50	67	70/87	0.80	No	No	No	1	<3	spicy	large
22	3661/05	60	F	housewife	Chennai	140	50	25.51	82	85/99	0.86	No	No	No	1	<3	spicy	moderate
23	3916/05	33	F	housewife	Chennai	149	54	24.32	77	77/93	0.83	No	No	No	3	>3	spicy	moderate
24	4789/05	48	F	maid	Chennai	150	47	20.89	83	85/95	0.89	No	No	No	2	<3	spicy	moderate
25	Fathima	50	F	housewife	Chennai	147	60	27.77	80.5	87/92	0.94	No	No	No	>3	>3	spicy	moderate
26	713/06	45	M	security	Chennai	155	52	21.64	76	82/93	0.88	No	No	No	1	1	spicy	moderate
27	847/06	42	F	housewife	Chennai	146	49	23.00	81	82/90	0.91	No	No	No	3	3	spicy	moderate
28	972/06	54	M	labourer	Chennai	166	53	19.23	80	83/89	0.93	Yes	No	No	1	0	spicy	moderate
29	1397/06	40	F	housewife	Chennai	149	42	18.92	64	67/87	0.77	No	No	No	1		spicy	small
30	1780/06	50	F	housewife	Chennai	150	57	25.33	83	85/102	0.80	No	No	No	2	2	spicy	moderate
31	2331/06	37	F	labourer	Chennai	168	66	23.38	83	86/94	0.90	No	No	No	2	<3	spicy	moderate
32	2470/06	60	F	housewife	Chennai	152	40	17.31	66	65/90	0.70	No	No	No	1	0	spicy	moderate
33	3036/06	54	M	marketing	Chennai	158	61	24.43	91	92/99	0.90	No	No	No	1	>3	spicy	moderate
34	3090/06	42	M	labourer	Chennai	164	52	19.33	76	78/83	0.94	Yes	No	Yes	3	<3	spicy	moderate
35	4340/06	39	M	labourer	Chennai	178	61	19.25	76	76/96	0.79	Yes	Yes	Yes	3	3	spicy	large
36	4829/06	40	F	housewife	Arcot	157	62	25.15	84	83/106	0.78	No	No	No	0	0	spicy	moderate
37	5200/06	29	M	driver	Chennai	168	53	18.79	72	75/85	0.88	No	No	Yes	>3	>3	spicy	moderate
38	5248/06	50	M	labourer	Chennai	169	66	23.11	85	87/96	0.91	Yes	Yes	Yes	3	>3	spicy	large
39	5284/06	50	F	vendor	Chennai	145	42	19.97	84	74/93	0.79	No	No	No	1	>3	spicy	moderate
40	5293/06	33	F	maid	Chennai	157	60	24.34	87	72/102	0.90	No	No	No	1	<3	spicy	moderate
41	6224/06	44	F	housewife	Chennai	155	76	31.63	94	93/110	0.84	No	No	No	1	<3	spicy	large
42	6294/06	39	F	housewife	Chennai	147	34	15.73	63	64/75	0.85	No	No	No	1	>3	spicy	small
43	6352/06	32	M	driver	Chennai	170	75	25.95	88	90/100	0.90	No	No	Yes	3	<3	spicy	moderate

Sl.No	MGE No	Age	Gender	Occupation	Place	Height	Weight	BMI	abd girth	waist/hip	W-H ratio	Smoking	Tobacco	Alcohol	NV/Week	friedfood/w	quality	quantity
44	6911/06	35	F	labourer	Chennai	160	59	23.05	82	85/100	0.85	No	No	No	1	<3	spicy	moderate
45	7072/06	45	F	cowherd	Chennai	160	46	17.97	67	70/93	0.75	No	No	No	1	>3	spicy	large
46	7182/06	65	F	housewife	Chennai	162	69	26.29	94	91/104	0.87	No	No	No	1	0	spicy	large
47	210/07	70	M	-	Chennai	170	67	23.18	93	93/99	0.94	No	Yes	No	2	>3	spicy	large
48	287/07	45	F	housewife	Chennai	166	47	17.06	68	66.5/99	0.67	No	No	No	2	>3	spicy	moderate
49	1108/07	60	M	security	Chennai	164	72	26.77	102	97/104	0.90	Yes	Yes	Yes	3	0	spicy	moderate
50	1175/07	43	F	housewife	Chennai	154	50	21.08	75	74/91	0.81	No	No	No	3	<3	spicy	moderate
51	1776/07	46	F	housewife	Chennai	156	51	20.96	77	82/90	0.90	No	Yes	No	2	3	spicy	large
52	1958/07	28	M	clerk	Chennai	163	42	15.81	60	61/75	0.80	Yes	No	No	1	0	spicy	moderate
53	1802/07	63	F	housewife	Chennai	154	56	23.61	85	86/102	0.80	No	Yes	No	3	<3	spicy	moderate
54	1830/07	57	M	clerk	Chennai	170	56	19.38	84	83/93	0.89	No	No	No	0	<3	spicy	moderate
55	1871/07	30	M	labourer	Chennai	175	66	21.55	84	87/98	0.89	No	No	No	1	>3	spicy	moderate
56	2121/07	38	F	housewife	Chennai	158	45	18.02	74	73/87	0.84	No	No	No	2	2	spicy	moderate
57	2392/07	51	M	labourer	Chennai	165	63	23.14	84	83/93	0.89	Yes	No	Yes	1	0	spicy	moderate
58	3036/07	45	M	labourer	Chennai	168	52	18.42	69	69/85	0.80	No	No	No	2	0	spicy	moderate
59	3081/07	32	M	labourer	Chennai	164	52	19.33	71	77/86	0.89	No	Yes	Yes	>3	>3	spicy	large
60	3142/07	23	F	marketing	Chennai	150	54	24.00	84	81/95	0.85	No	No	No	1	1	spicy	moderate
61	3213/07	70	M	-	Chennai	164	65	23.30	97	94/108	0.87	No	No	No	3	<3	spicy	moderate
62	3217/07	50	F	housewife	Chennai	155	68	28.30	98	99/107	0.89	No	No	No	1	0	spicy	moderate
63	3434/07	44	F	housewife	Chennai	160	70	27.34	95	99/109	0.90	No	No	No	2	,3	spicy	moderate
64	3477/07	41	M	clerk	Chennai	167	53	19.00	82	83/91	0.90	Yes	Yes	Yes	3	>3	spicy	moderate
65	3597/07	45	F	housewife	Chennai	148	57	26.02	82	84/104	0.80	No	No	No	1	0	spicy	small
66	3958/07	61	M	-	Chennai	174	62	20.48	85	88/95	0.90	No	No	Yes	2	0	spicy	moderate
67	4109/07	55	M	-	Chennai	167	59	21.16	87	92/94	0.97	No	Yes	Yes	1	<3	spicy	moderate
68	4596/07	38	M	security	Chennai	166	60	21.77	87	88/96	0.90	No	No	No	1	0	spicy	large
69	/07	60	F	housewife	Chennai	143	53	25.92	86	88/101	0.87	No	No	No	2	>3	spicy	moderate
70	4983/07	36	M	labourer	Chennai	170	70	24.22	88	95/100	0.95	No	No	No	2	<3	spicy	large
71	5012/07	50	F	housewife	Chennai	152	76	32.68	103	104/117	0.89	No	No	No	2	2	spicy	moderate
72	5072/07	48	F	housewife	Chennai	150	52	23.11	76	71/91	0.78	No	No	No	2	>3	spicy	moderate
73	5318/07	75	M	-	Chennai	164	56	20.82	82	80/91	0.88	Yes	No	No	1	0	spicy	moderate
74	6133/07	35	M	marketing	Chennai	160	66	25.78	89	93/102	0.90	Yes	Yes	Yes	3	>3	spicy	moderate
75	6142/07	59	F	housewife	Chennai	154	62	26.14	92	98/105	0.90	No	No	No	2	1	spicy	moderate
76	6194/07	45	F	housewife	Chennai	155	51	21.23	80	82/98	0.80	No	No	No	1	>3	spicy	moderate
77	6480/07	65	F	housewife	Chennai	134	34	18.93	68	73/85	0.86	No	No	No	2	2	spicy	moderate
78	6760/07	45	M	labourer	Chennai	164	65	24.17	84	86/96	0.89	Yes	No	No	3	3	spicy	large
79	6875/07	50	F	housewife	Chennai	149	65	29.28	89	88/108	0.80	No	No	No	2	1	spicy	large
80	7066/07	56	M	-	Chennai	165	50	18.36	74	78/84	0.90	Yes	No	No	1	1	spicy	large
81	7195/07	40	M	labourer	Chennai	169	54	18.91	74	80/87	0.90	No	No	No	3	<3	spicy	moderate
82	52/08	60	F	maid	Chennai	149	52	23.42	79	80/96	0.80	No	Yes	No	1	<3	spicy	moderate
83	342/08	47	F	housewife	Chennai	156	64	26.29	91	89/104	0.85	No	Yes	No	4	>3	spicy	large
84	361/08	29	F	maid	Chennai	156	62	25.47	96	86/103	0.80	No	No	No	3	3	spicy	large
85	554/08	45	M	labourer	Chennai	162	57	21.72	81	84/94	0.89	Yes	No	Yes	7	>3	spicy	large
86	907/08	55	F	labourer	Chennai	155	85	35.38	100	105/120	0.87	No	No	No	1	0	spicy	moderate
87	927/08	50	F	housewife	Chennai	150	60	26.66	78	78/100	0.78	No	No	No	1	1	spicy	large
88	1270/08	21	M	vendor	Chennai	169	54	18.91	65	69/86	0.80	No	No	Yes	1	<3	spicy	large

Sl.No	MGE No	Age	Gender	Occupation	Place	Height	Weight	BMI	abd girth	waist/hip	W-H ratio	Smoking	Tobacco	Alcohol	NV/Week	friedfood/w	quality	quantity
89	1478/08	53	F	maid	Chennai	153	56	23.92	78	85/95	0.89	No	No	No	1	>3	spicy	large
90	1738/08	38	F	labourer	Chennai	154	67	28.25	86	94/113	0.80	No	Yes	No	>3	>3	spicy	large
91	1910/08	28	F	housewife	Chennai	157	50	20.28	72	74/89	0.80	No	No	No	1	>3	spicy	moderate
92	2209/08	65	M	-	Chennai	168	56	19.84	84	86/90	0.98	Yes	No	No	1	0	spicy	large
93	2282/08	43	F	housewife	Chennai	144	48	23.15	76	76/96	0.79	No	No	No	2	3	spicy	moderate
94	2455/08	28	F	housewife	Chennai	155	50	20.81	74	74/89	0.80	No	No	No	1	0	spicy	large
95	2640/08	40	M	labourer	Chennai	170	60	20.76	83	85/92	0.90	Yes	Yes	Yes	>3	>3	spicy	large
96	2814/08	20	M	-	Chennai	170	62	21.45	70	72/88	0.80	No	No	No	1	1	spicy	large
97	2868/08	40	F	labourer	Chennai	150	59	26.22	84	85/100	0.85	No	No	No	1	<1	spicy	moderate
98	2927/08	40	M	labourer	Chennai	168	47	27.98	65	65/79	0.80	Yes	No	No	1	0	spicy	moderate
99	2956/08	47	F	housewife	Chennai	157	67	27.18	85	85/102	0.80	No	No	No	1	0	spicy	moderate
100	3112/08	52	M	security	Chennai	157	71	28.80	100	100/97	1.03	No	No	No	1	1	spicy	large
101	3565/08	23	M	driver	Chennai	175	51	16.65	67	68/78	0.87	Yes	Yes	No	1	1	spicy	moderate
102	62588	62	F	housewife	Chennai	150	48	21.33	80	85/97	0.88	No	No	No	2	0	spicy	large
103	108/08	55	M	labourer	Chennai	170	63	21.79	87	90/97	0.90	Yes	No	No	2	2	spicy	moderate
104	3353/08	44	M	labourer	Chennai	155	40	16.65	63	67/89	0.80	Yes	Yes	Yes	3	3	spicy	moderate
105	2653/08	62	M	-	Chennai	165	49	17.99	72	74/85	0.87	No	No	No	2	>3	spicy	moderate
106	3355/08	47	F	labourer	Chennai	153	36	15.38	61	59/78	0.76	No	No	No	3	3	spicy	moderate

Sl.No	citrusfruit	fat	chocolate	coffee/tea	softdrink	NSAID	Pill	infection	radiation	pregnant	NG aspir	Scleroderm	Heartburn	duration	D/N/Both	D/occ/rare	Regurg	duration	D/N/Both	D/W/M
1	Yes	No	Yes	1	Yes	Yes	No	No	No	No	No	No	Yes	6 months	D	D	Yes	6 monnths	D	D
2	No	Yes	No	2	No	No	No	No	No	No	No	No	Yes	3 months	D	D	Yes	3 months	D	D
3	No	No	No	3	Yes	Yes	No	No	No	No	No	No	Yes	6 yrs	D	rare	Yes	6 yrs	D	M
4	Yes	Yes	Yes	2	Yes	No	No	No	No	No	No	No	No				Yes	3 yrs	D	W
5	Yes	No	No	2	No	Yes	No	No	No	No	No	No	Yes	1 yr	Both	D	Yes	1 yr	Both	D
6	Yes	Yes	No	4	Yes	Yes	No	No	No	No	No	No	Yes	5 yrs	Both	D	Yes	5 yrs	D	D
7	Yes	Yes	No	3	No	No	No	No	No	No	No	No	No				Yes	5 months	Both	W
8	Yes	No	No	2	Yes	Yes	No	No	No	No	No	No	No				Yes	6 yrs	D	W
9	Yes	Yes	No	2	Yes	Yes	No	No	No	No	No	No	Yes	4 yrs	D	occ	Yes	4 yrs	D	D
10	Yes	No	Yes	4	Yes	No	No	Yes	No	No	No	No	Yes	2 yrs	D	D	Yes	2 yrs	D	D
11	Yes	Yes	No	2	Yes	No	No	No	No	No	No	No	Yes	4 yrs	Both	D	Yes	4 yrs	Both	D
12	Yes	No	No	1	No	No	No	No	No	No	No	No	Yes	4 yrs	N	D	No			
13	No	No	No	6	Yes	Yes	No	No	No	No	No	No	Yes	4 yrs	D	D	No			
14	Yes	Yes	Yes	3	No	No	No	No	No	No	No	No	Yes	10 days	D	D	Yes	10 days	D	D
15	No	No	No	2	Yes	No	No	No	No	No	No	No	Yes	4 yrs	Both	D	Yes	4 yrs	Both	D
16	No	Yes	No	2	Yes	Yes	No	No	No	No	No	No	Yes	4 yrs	Both	D	Yes	4 yrs	Both	D
17	Yes	Yes	No	2	No	No	No	No	No	No	Yes	No	Yes	3 yrs	Both	D	Yes	3 yrs	Both	D
18	No	No	No	2	No	No	No	No	No	No	No	No	Yes	5 yrs	Both	D	No			
19	No	No	No	2	No	No	No	No	No	No	No	No	Yes	4 months	Both	D	No			
20	Yes	Yes	No	5	Yes	Yes	No	No	No	No	No	No	Yes	2.5 yrs	Both	D	Yes	2.5 yrs	Both	D
21	No	No	No	1	No	Yes	No	No	No	No	No	No	No				Yes	3 months	Both	D
22	Yes	No	Yes	2	Yes	Yes	No	No	No	No	No	No	Yes	1 month	D	occ	Yes	1 month	Both	D
23	No	Yes	Yes	2	Yes	Yes	No	No	No	No	No	No	Yes	3 yrs	Both	D	Yes	3 yrs	D	D
24	Yes	No	No	3	Yes	Yes	No	No	No	No	No	No	Yes	3.5 yrs	Both	occ	Yes	3.5 yrs	Both	W
25	No	Yes	No	3	No	No	No	No	No	No	No	No	Yes	5 yrs	Both	D	Yes	5 yrs	Both	D
26	Yes	Yes	No	2	Yes	Yes	No	No	No	No	No	No	Yes	3 yrs	D	D	Yes	3 yrs	D	D
27	No	Yes	No	2	Yes	Yes	No	No	No	No	No	No	Yes	2 months	Both	D	Yes	2 months	Both	D
28	No	No	No	10	No	No	No	No	No	No	No	No	Yes	1 month	D	D	Yes	1 month	D	D
29	Yes	No	No	4	Yes	No	No	No	No	No	No	No	Yes	5 yrs	Both	occ	Yes	3 months	Both	W
30	No	Yes	No	0	Yes	No	No	No	No	No	No	No	Yes	2 yrs	D	D	Yes	2 yrs	D	D
31	Yes	Yes	No	4	Yes	Yes	No	No	No	No	No	No	Yes	2.5 yrs	Both	occ	Yes	6 monnths	D	W
32	Yes	No	No	0	No	No	No	No	No	No	No	No	Yes	5 yrs	Both	D	No			
33	Yes	Yes	Yes	4	Yes	No	No	No	No	No	No	No	Yes	2 yrs	Both	D	Yes	2 yrs	N	D
34	Yes	Yes	No	10	No	No	No	No	No	No	No	No	Yes	1.5 months	D	D	No			
35	Yes	No	No	1	Yes	Yes	No	No	No	No	No	No	Yes	3 yrs	Both	D	Yes	3 yrs	Both	D
36	No	No	No	3	No	Yes	No	No	No	No	No	No	Yes	2.5 yrs	D	occ	Yes	2.5 yrs	D	W
37	Yes	Yes	No	3	Yes	Yes	No	No	No	No	No	No	Yes	1 yr	D	D	Yes	1 yr	Both	D
38	Yes	Yes	Yes	2	Yes	No	No	No	No	No	No	No	Yes	3 months	D	D	Yes	3 months	D	D
39	No	No	Yes	2	No	No	No	No	No	No	No	No	Yes	2 yrs	Both	rare	Yes	2yrs	D	M
40	No	No	No	1	Yes	No	No	No	No	No	No	No	No				Yes	2 yrs	D	M
41	Yes	Yes	No	2	No	Yes	No	No	No	No	No	No	Yes	1.5 yrs	D	occ	No			
42	Yes	No	No	3	Yes	Yes	y	No	No	No	No	No	Yes	3 yrs	D	D	Yes	3 months	D	W
43	Yes	No	No	1	No	No	No	No	No	No	No	No	Yes	2 yrs	D	D	Yes	2 yrs	D	D

Sl.No	citrusfruit	fat	chocolate	coffee/tea	softdrink	NSAID	Pill	infection	radiation	pregnant	NG aspir	Scleroderm	Heartburn	duration	D/N/Both	D/occ/rare	Regurg	duration	D/N/Both	D/W/M
44	Yes	Yes	No	2	Yes	Yes	No	No	No	No	No	No	Yes	3 months	Both	D	Yes	3 months	Both	D
45	No	No	No	2	Yes	Yes	No	No	No	No	No	No	Yes	1 yr	Both	D	Yes	1 yr	D	D
46	No	No	No	2	No	Yes	No	No	No	No	No	No	No				Yes	1 yr	Both	D
47	Yes	Yes	Yes	3	Yes	Yes	No	No	No	No	No	No	Yes	2 yrs	Both	D	No			
48	No	Yes	No	5	No	No	No	No	No	No	No	No	Yes	1 yr	D	rare	Yes	1 yr	D	W
49	No	No	No	3	No	Yes	No	No	No	No	No	No	Yes	2 yrs	D	occ	No			
50	No	No	Yes	3	No	Yes	No	No	No	No	No	No	Yes	2 months	D	D	Yes	2 months	night	D
51	No	No	No	2	No	Yes	No	No	No	No	No	No	Yes	3 months	Both	D	Yes	3 months	Both	D
52	Yes	No	No	3	No	No	No	No	No	No	No	No	Yes	3 months	Both	D	Yes	3 months	Both	D
53	No	No	No	4	Yes	Yes	No	No	No	No	No	No	Yes	2.5 yrs	D	D	Yes	2.5 yrs	D	D
54	Yes	No	No	3	Yes	Yes	No	No	No	No	No	No	Yes	1.5 yrs	D	D	Yes	1.5 yrs	D	D
55	No	No	Yes	0	Yes	Yes	No	No	No	No	No	No	Yes	1.5 yrs	Both	D	Yes	1 yr	Both	D
56	No	No	No	2	No	Yes	No	No	No	No	No	No	Yes	2 yrs	D	D	Yes	2 yrs	D	W
57	Yes	Yes	Yes	3	No	No	No	No	No	No	No	No	Yes	3 yrs	Both	D	Yes	3 yrs	Both	D
58	Yes	No	No	2	No	Yes	No	No	No	No	No	No	Yes	3 months	D	D	Yes	3 months	D	D
59	Yes	No	Yes	5	No	Yes	No	No	No	No	No	No	No				Yes	1 yr	D	W
60	Yes	Yes	Yes	0	Yes	Yes	No	No	No	No	No	No	Yes	2 yrs	Both	D	Yes	2 yrs	D	D
61	No	Yes	No	2	No	No	No	No	No	No	No	No	Yes	1.5 yrs	N	D	Yes	1.5 yrs	Both	D
62	Yes	No	No	0	Yes	Yes	No	No	No	No	No	No	Yes	1.5 yrs	Both	W	Yes	1.5 yrs	D	D
63	Yes	Yes	No	2	No	Yes	No	No	No	No	No	No	Yes	1 yr	Both	D	Yes	1 yr	D	D
64	Yes	Yes	Yes	0	No	Yes	No	No	No	No	No	No	Yes	8 months	D	D	No			
65	No	No	No	2	Yes	No	No	No	No	No	No	No	Yes	1.5 yrs	D	D	Yes	1.5 yrs	Both	D
66	Yes	No	No	4	No	Yes	No	No	No	No	No	No	Yes	4 yrs	N	D	Yes	4 yrs	N	D
67	Yes	Yes	No	8	Yes	Yes	No	No	No	No	No	No	Yes	2 yrs	Both	D	Yes	2 yrs	Both	D
68	Yes	No	No	2	Yes	Yes	No	No	No	No	No	No	Yes	2.5 yrs	Both	W	Yes	2.5 yrs	Both	W
69	Yes	Yes	Yes	2	Yes	Yes	No	No	No	No	No	No	No				Yes	7 months	D	D
70	Yes	No	No	3	No	Yes	No	No	No	No	No	No	Yes	3 months	N	W	No			
71	Yes	Yes	Yes	1	Yes	Yes	No	No	No	No	No	No	Yes	1 yr	Both	W	Yes	1 yr	Both	W
72	No	Yes	Yes	3	Yes	No	No	No	No	No	No	No	Yes	2 yrs	D	D	Yes	2 yrs	D	D
73	Yes	No	No	6	No	No	No	No	No	No	No	No	Yes	5 yrs	Both	D	Yes	5 yrs	Both	D
74	No	Yes	No	0	Yes	No	No	No	No	No	No	No	Yes	5 yrs	Both	D	No			
75	No	No	No	0	No	Yes	No	No	No	Yes	No	No	Yes	6 months	D	D	Yes	6 monnths	D	D
76	No	No	No	0	No	No	No	No	No	No	No	No	No				No			
77	No	No	No	2	No	Yes	No	No	No	No	No	No	Yes	1 yr	D	D	Yes	1 yr	D	D
78	Yes	Yes	No	0	Yes	No	No	No	No	No	No	No	Yes	3 yrs	D	D	No			
79	Yes	Yes	Yes	3	No	Yes	No	No	No	No	No	No	Yes	6 months	D	D	Yes	6 monnths	Both	D
80	Yes	No	No	2	No	Yes	No	No	No	No	No	No	Yes	6 months	Both	D	Yes	6 monnths	Both	D
81	Yes	Yes	No	4	Yes	No	No	No	No	No	No	No	Yes	2 months	D	W	No			
82	No	No	No	3	No	Yes	No	No	No	No	No	No	Yes	6 months	D	D	No			
83	Yes	Yes	Yes	3	No	Yes	No	No	No	No	No	No	Yes	2 months	Both	W	No			
84	No	No	Yes	3	No	Yes	Yes	No	No	No	No	No	No				No			
85	No	Yes	No	3	No	Yes	No	No	No	No	No	No	No				Yes	2 months	Both	D
86	No	No	No	8	Yes	Yes	No	No	No	No	No	No	Yes	1 month	Both	D	Yes	1 month	D	D
87	No	No	Yes	1	Yes	Yes	No	No	No	No	No	No	Yes	5 yrs	D	D	No			
88	No	Yes	Yes	2	Yes	No	No	No	No	No	No	No	No				No	5 months	D	D

Sl.No	citrusfruit	fat	chocolate	coffee/tea	softdrink	NSAID	Pill	infection	radiation	pregnant	NG aspir	Scleroderm	Heartburn	duration	D/N/Both	D/occ/rare	Regurg	duration	D/N/Both	D/W/M
89	No	Yes	Yes	2	No	No	No	No	No	No	No	No	Yes	5 months	Both	D	Yes	5 months	D	M
90	No	Yes	Yes	6	No	Yes	No	No	No	No	No	No	No				Yes	3 months	Both	W
91	Yes	Yes	No	3	No	Yes	Yes	No	No	No	No	No	Yes	3 months	D	W	Yes	3 months	D	W
92	No	No	No	2	No	No	No	No	No	No	No	No	No				Yes	4 months	D	D
93	No	No	No	3	No	No	No	No	No	No	No	No	Yes	2 yrs	D	D	Yes	2 yrs	D	D
94	Yes	No	No	1	Yes	Yes	No	No	No	No	No	No	Yes	1 yr	D	D	Yes	1 yr	D	D
95	Yes	Yes	No	8	Yes	Yes	No	No	No	No	No	No	Yes	1 yr	D	D	Yes	5 yrs	Both	D
96	Yes	Yes	Yes	2	Yes	Yes	No	No	No	No	No	No	Yes	1 month	N	W	No			
97	No	Yes	No	2	No	Yes	No	No	No	No	No	No	Yes	8 months	D	D	Yes	8 months	D	D
98	Yes	No	No	2	No	Yes	No	No	No	No	No	No	Yes	6 months	D	D	Yes	6 monnths	D	D
99	No	No	No	2	No	No	No	No	No	No	No	No	Yes	1 month	Both	D	Yes	1 month	Both	D
100	Yes	Yes	No	2	Yes	No	No	No	No	No	No	No	Yes	2 yrs	D	W	Yes	2 yrs	D	W
101	No	Yes	Yes	6	No	Yes	No	No	No	No	No	No	Yes	2 weeks	Both	D	Yes	2 week	D	D
102	No	Yes	No	4	No	Yes	No	No	No	No	No	No	Yes	8 yrs	Both	D	Yes	8 yrs	Both	D
103	Yes	No	No	2	No	No	No	No	No	No	No	No	No				Yes	1 yr	D	W
104	Yes	Yes	Yes	5	Yes	No	No	No	No	No	No	No	No				Yes	10 days	Both	D
105	Yes	No	No	2	No	No	No	No	No	No	No	No	Yes	1.5 yrs	Both	W	No			
106	Yes	Yes	No	1	No	Yes	No	No	No	No	No	No	Yes	3 months	Both	D	Yes	3 months	D	D

Sl.No	bending	postmeal	Dysphagia	int/cont	sol/liq/Both	Chestpain	duration	severity	postmeal	waterbrash	odyNophag	burping	bloating	satiety	hiccup	nausea	vomiting	asthma
1	Increase	Increase	No			No				No	No	No	No	No	No	No	No	No
2	Increase	Increase	No			No				No	No	Yes	Yes	Yes	No	No	No	No
3		Increase	No			Yes	6 yrs	moderate		No	No	Yes	Yes	Yes	No	Yes	Yes	No
4		Increase	No			No				No	No	Yes	No	No	No	No	No	No
5	Increase	Increase	Yes	int	sol	No				Yes	No	Yes	No	Yes	No	Yes	Yes	No
6		Increase	Yes	int	Both	No				No	No	No	Yes	Yes	Yes	Yes	Yes	No
7			Yes	int	sol	No				Yes	No	No	No	No	No	No	No	No
8	Increase		No			No				No	No	No	No	No	No	Yes	No	No
9	Increase	Increase	No			No				No	No	Yes	Yes	Yes	No	No	No	No
10	Increase		Yes	cont	sol	No				No	No	Yes	No	Yes	No	No	No	No
11	Increase	Increase	No			No				No	No	No	Yes	Yes	No	No	No	No
12			No			No				No	No	No	No	No	No	No	No	No
13			No			No				No	No	No	Yes	Yes	No	No	No	No
14	Increase	Increase	No			Yes	10 days	mild	Increase	No	No	No	Yes	Yes	No	Yes	No	No
15	Increase	Increase	No			No				Yes	No	No	Yes	Yes	No	Yes	Yes	No
16	Increase	Increase	Yes	int	sol	No				No	No	No	Yes	Yes	No	Yes	Yes	No
17	Increase	Increase	No			No				Yes	No	Yes	Yes	Yes	No	No	No	No
18			No			No				No	No	No	Yes	No	No	No	Yes	No
19			Yes	int	sol	Yes	4 months	moderate		No	No	No	No	No	No	No	No	No
20	Increase	Increase	No			No				No	No	No	Yes	No	No	No	No	No
21	Increase		No			No				No	No	Yes	Yes	Yes	No	Yes	No	No
22		Increase	No			Yes	1 month	mild		No	No	No	No	Yes	Yes	No	No	No
23	Increase	Increase	No			Yes	3 yrs	moderate		No	Yes	Yes	Yes	Yes	No	Yes	Yes	No
24	Increase	Increase	Yes	int	sol	Yes	8 yrs	moderate		No	No	Yes	Yes	Yes	Yes	Yes	Yes	Yes
25		Increase	No							Yes	No	Yes	Yes	Yes	No	Yes	Yes	Yes
26	Increase	Increase	Yes	int	sol	No				Yes	No	Yes	Yes	Yes	Yes	Yes	No	No
27		Increase	No			Yes	2 months	moderate	Increase	Yes	No	Yes	Yes	Yes	No	No	No	No
28		Increase	No			No				No	No	No	Yes	Yes	No	No	No	No
29		Increase	No			Yes	3 months	moderate	Increase	No	No	No	No	No	No	No	No	No
30	Increase	Increase	No			No				No	No	Yes	No	No	No	Yes	No	No
31		Increase	No			Yes	6 months	mild		No	No	Yes	No	No	No	No	No	No
32			Yes	int	sol	No				No	No	No	Yes	No	No	No	No	No
33			No			Yes	2 yrs	mild	Increase	Yes	No	Yes	Yes	Yes	No	No	No	No
34			No			No				No	No	No	Yes	Yes	No	No	No	No
35		Increase	Yes	int	liquids	No				Yes	No	No	Yes	No	No	Yes	No	No
36	Increase	Increase	No			No				No	No	No	No	No	No	Yes	Yes	No
37	Increase	Increase	Yes	int	sol	No				Yes	No	No	No	No	No	No	No	No
38		Increase	No			Yes	3 months	mild	Increase	No	No	No	Yes	Yes	No	Yes	Yes	No
39			No			No				No	No	No	No	No	No	Yes	Yes	No
40	Increase	Increase	No			No				No	No	No	No	No	No	No	No	No
41			No			Yes	2 yrs	moderate	Increase	No	No	No	Yes	Yes	No	No	No	Yes
42	Increase		No			No				No	No	No	Yes	Yes	No	No	No	No
43			No			No				Yes	No	Yes	Yes	No	No	No	No	No

Sl.No	bending	postmeal	Dysphagia	int/cont	sol/liq/Both	Chestpain	duration	severity	postmeal	waterbrash	odyNophag	burping	bloating	satiety	hiccup	nausea	vomiting	asthma
44	Increase	Increase	Yes	int	sol	No				No	No	Yes	Yes	Yes	No	No	No	No
45	Increase	Increase	Yes	int	sol	Yes	1 yr	mild		Yes	No	No	No	Yes	No	No	No	No
46	Increase	Increase	No			No				No	No	Yes	Yes	Yes	No	No	No	No
47			Yes	int	sol	Yes	2 yrs	moderate	Increase	No	No	No	Yes	No	No	No	No	No
48	Increase		No			Yes	1 yr	mild		Yes	No	Yes	No	Yes	No	No	No	No
49			No			No				No	No	Yes	No	No	No	No	No	No
50	Increase	Increase	No			No				No	No	Yes	Yes	Yes	No	Yes	Yes	No
51	Increase	Increase	No			Yes	2 weeks	mild		Yes	No	Yes	Yes	Yes	Yes	Yes	Yes	No
52	Increase	Increase	No			No				No	No	No	Yes	Yes	No	Yes	Yes	No
53	Increase		No			No				Yes	No	No	No	No	No	No	No	No
54	Increase	Increase	Yes	cont	sol	Yes	3 months	moderate	Increase	No	Yes	Yes	Yes	Yes	No	Yes	No	No
55	Increase	Increase	Yes	int	liquids	No				Yes	Yes	No	Yes	Yes	No	No	No	No
56		Increase	No			No				No	No	No	No	No	No	No	No	No
57	Increase	Increase	Yes	int	sol	Yes	3 yrs	moderate		Yes	No	Yes	No	No	Yes	No	No	No
58	Increase	Increase	No			No				No	No	No	No	Yes	No	No	No	No
59	Increase	Increase	Yes	int	Both	No				No	No	Yes	Yes	Yes	No	No	No	Yes
60		Increase	Yes	int	sol	Yes	2 yrs	moderate		No	No	No	Yes	Yes	No	Yes	Yes	No
61		Increase	Yes	int	sol	No				Yes	No	No	Yes	Yes	No	No	Yes	No
62	Increase		No			No				No	No	Yes	Yes	Yes	No	Yes	Yes	No
63	Increase	Increase	No			Yes	1 yr	moderate		No	No	Yes	Yes	Yes	No	No	No	No
64			No			No				No	No	Yes	No	No	No	Yes	Yes	No
65	Increase	Increase	Yes	int	sol	Yes	1.5 yrs	moderate		Yes	No	No	Yes	Yes	Yes	Yes	Yes	Yes
66	Increase		No			No				No	No	No	No	No	No	No	No	No
67		Increase	Yes	int	sol	No				Yes	No	Yes	Yes	Yes	No	Yes	No	No
68	Increase	Increase	Yes	int	sol	Yes	2 months	moderate	Increase	No	No	Yes	Yes	Yes	No	Yes	No	Yes
69	Increase	Increase	Yes	cont	Both	No				No	No	Yes	Yes	Yes	No	No	No	No
70			No			No				No	No	No	No	No	Yes	No	No	No
71		Increase	Yes	int	Both	No				No	Yes	Yes	Yes	No	No	No	No	No
72		Increase	No			No				No	No	No	Yes	Yes	No	Yes	No	No
73	Increase		No			No				Yes	No	No	No	No	No	No	No	No
74			No			No				No	No	No	Yes	Yes	No	Yes	Yes	No
75	Increase	Increase	Yes	int	sol	Yes	6 months	moderate		Yes	No	No	No	No	No	Yes	No	No
76			No			Yes	1 month	severe	Increase	Yes	No	Yes	Yes	Yes	Yes	Yes	No	No
77	Increase	Increase	Yes	int	sol	Yes	1 yr	moderate	Increase	No	No	Yes	No	No	Yes	No	No	No
78			No			No				Yes	No	No	Yes	Yes	No	No	No	No
79	Increase	Increase	Yes	int	sol	No				Yes	No	Yes	Yes	Yes	Yes	No	No	No
80	Increase	Increase	Yes	int	sol	No				Yes	No	Yes	Yes	Yes	Yes	No	Yes	Yes
81			No			Yes	1 week	mild		No	No	Yes	Yes	Yes	Yes	No	No	No
82			Yes	int	sol	Yes	5 yrs	mild	Increase	Yes	No	No	No	No	Yes	No	No	No
83			No			Yes	3 yrs	moderate		No	No	Yes	No	No	No	Yes	No	No
84			No			Yes	5 yrs	mild	Increase	No	No	Yes	No	No	No	No	No	No
85	Increase	Increase	No			Yes	2 months	severe	Increase	No	No	Yes	No	No	No	No	No	No
86	Increase	Increase	No			Yes	1 month	mild	Increase	Yes	No	No	Yes	Yes	No	No	No	No
87			No			No				No	No	No	No	No	No	No	No	No
88	Increase	Increase	Yes	int	sol	No				No	No	No	Yes	Yes	Yes	No	Yes	No

Sl.No	bending	postmeal	Dysphagia	int/cont	sol/liq/Both	Chestpain	duration	severity	postmeal	waterbrash	odyNophag	burping	bloating	satiety	hiccup	nausea	vomiting	asthma
89	Increase	Increase	No			No				Yes	No	Yes	No	No	Yes	No	No	No
90	Increase		No			Yes	3 months	mild	Increase	No	No	Yes	Yes	Yes	No	No	No	No
91	Increase	Increase	No			No				No	No	No	No	No	No	No	No	No
92		Increase	No			No				No	No	No	No	No	No	No	No	No
93	Increase	Increase	No			No				No	No	No	No	No	No	No	No	No
94	Increase	Increase	Yes	int	sol	No				No	No	No	Yes	Yes	No	Yes	No	No
95	Increase	Increase	No			No				No	No	Yes	Yes	Yes	Yes	Yes	Yes	No
96			Yes	int	Both	No				No	Yes	No	No	No	Yes	No	No	No
97	Increase	Increase	No			No				No	No	Yes	Yes	Yes	No	Yes	No	No
98	Increase	Increase	No			No				No	No	No	Yes	Yes	No	No	No	Yes
99	Increase	Increase	No			Yes	1 month	mild	Increase	No	No	No	No	No	No	No	No	No
100			Yes	cont	sol	No				No	Yes	Yes	Yes	Yes	No	No	No	Yes
101	Increase	Increase	Yes	int	sol	No				No	No	No	Yes	Yes	No	No	No	No
102		Increase	Yes	int	sol	Yes	2 months	mild		No	No	No	No	No	No	No	Yes	No
103		Increase	Yes	int	sol	No				Yes	No	Yes	Yes	Yes	Yes	No	No	No
104		Increase	Yes	int	sol	No				No	No	Yes	No	No	No	No	No	No
105			No			No				No	No	No	Yes	Yes	No	No	No	No
106		Increase	No			No				No	No	Yes	Yes	Yes	No	No	No	No

Sl.No	hoarseness	chr cough	recLRI	caries	wt loss	UGI bleed	comorb	Scopy No	Normal	OeN/I/E/U	StN/I/E/U	DuN/I/E/U	HiatusH	Dietchange	L S mod	Resp to Rx	Rec
1	No	No	No	No	Yes	No		1219/02			U			Yes	No	Yes	Yes
2	No	No	No	Yes	No	No		825/03	N					Yes	No	Yes	Yes
3	No	No	No	No	No	No		252/03	N					Yes	No	Yes	Yes
4	No	No	No	Yes	No	No		1955/07		I		U		Yes	Yes	Yes	Yes
5	Yes	Yes	Yes	Yes	Yes	No	DM	3385/04				U	No	Yes	No	Yes	Yes
6	No	No	No	Yes	Yes	No		1376/03		N	N	I	No	Yes	No	Yes	Yes
7	No	No	No	No	Yes	No		2816/04		N	I	U	No	Yes	No	Yes	Yes
8	No	No	No	nn	No	No		2431/03	lax LES					Yes	No	Yes	Yes
9	No	No	No	Yes	No	No	Htn,DM	406/03	lax LES	I			Yes	Yes	Yes	Yes	Yes
10	No	No	No	Yes	No	No		4048/04				U	No	No	No	Yes	Yes
11	No	No	No	No	No	No	Htn	352/04					Yes	Yes	No	Yes	Yes
12	No	No	No	Yes	No	No		3543/04				U	No	Yes	Yes	Yes	Yes
13	No	No	No	Yes	No	No	trauma	2759/06						Yes	Yes	Yes	Yes
14	No	No	No	Yes	No	No		4128/04			E	E		Yes	No	Yes	Yes
15	No	No	No	No	Yes	Yes		4880/004	N					Yes	No	Yes	Yes
16	No	No	No	Yes	No	No		2029/05	N					Yes	No	Yes	Yes
17	Yes	No	No	No	No	No	Hypothyr	23/2006			E			Yes	No	Yes	Yes
18	No	No	No	No	No	No		633/05		I		I	Yes	Yes	No	Yes	Yes
19	No	No	No	No	No	No	DM	1475/05	N					No	No	Yes	Yes
20	Yes	Yes	Yes	No	No	No	Hypothyr	2005			I		Yes	Yes	No	Yes	Yes
21	No	No	No	Yes	Yes	No	Infertility	1674/05			I			Yes	No	Yes	Yes
22	Yes	Yes	Yes	No	No	No	DM/Htn	2102/05	N					Yes	No	Yes	Yes
23	No	No	No	Yes	No	No		2223/05		E	E			Yes	No	Yes	Yes
24	No	No	No	No	No	No		2698/05			U		Yes	Yes	No	Yes	Yes
25	No	Yes	No	No	Yes	No		3141/06	N					Yes	No	Yes	Yes
26	No	No	No	Yes	Yes	No	Hypoth,IH	621/06	N				No	Yes	No	Yes	Yes
27	No	No	Yes	No	No	No		2041/06	lax LES		I		Yes	Yes	No	Yes	Yes
28	No	No	No	No	No	No	Htn	49/08	lax LES					Yes	No	Yes	Yes
29	No	No	No	Yes	Yes	No		962/06	N					Yes	No	Yes	Yes
30	Yes	Yes	Yes	Yes	No	No		533/08		I,E				Yes	Yes	Yes	Yes
31	Yes	No	No	Yes	No	No		231/08	N					Yes	No	Yes	Yes
32	No	No	No	Yes	Yes	No		1379/06	N					Yes	No	Yes	Yes
33	No	No	No	No	No	No	DM/Htn	537/08		I				Yes	No	Yes	Yes
34	No	No	No	Yes	Yes	Yes		1777/06	lax LES			U		Yes	No	Yes	Yes
35	No	Yes	Yes	n	Yes	No		2308/06	N					Yes	No	Yes	Yes
36	No	No	No	Yes	No	No		1839/06	N					Yes	No	Yes	Yes
37	No	No	No	Yes	Yes	No		2597/06	lax LES					Yes	No	Yes	Yes
38	Yes	Yes	No	Yes	No	No		2576/06				E,U		Yes	No	Yes	Yes
39	No	No	No	Yes	Yes	No	DM	2660/06	N					No	No	Yes	Yes
40	No	No	No	No	No	No		2604/06			U	U		Yes	No	Yes	Yes
41	Yes	Yes	Yes	Yes	No	No			N					Yes	No	Yes	Yes
42	No	No	No	Yes	Yes	No		3000/06	N					Yes	No	Yes	Yes
43	No	No	No	No	No	No		967/07				U		Yes	No	Yes	Yes

Sl.No	hoarseness	chr cough	recLRI	caries	wt loss	UGI bleed	comorb	Scopy No	Normal	OeN/I/E/U	StN/I/E/U	DuN/I/E/U	HiatusH	Dietchange	L S mod	Resp to Rx	Rec
44	Yes	No	No	Yes	No	No		3927/06			I			Yes	No	Yes	Yes
45	No	No	No	Yes	Yes	No		3332/06	lax LES					Yes	No	Yes	Yes
46	No	No	No	Yes	No	No		160/07			E			Yes	Yes	Yes	Yes
47	No	No	No	Yes	No	No	Htn	221/07	bile gastr		I			Yes	Yes	Yes	Yes
48	No	No	No	No	No	No		150/07		N	U			No	No	Yes	Yes
49	No	No	No	No	No	No		589/07	N					Yes	No	Yes	Yes
50	No	No	No	Yes	No	No			N					Yes	No	Yes	Yes
51	No	No	No	Yes	No	No	DM	2390/07	N					Yes	No	Yes	Yes
52	No	No	No	No	No	No		944/07		I		I,U		Yes	No	Yes	Yes
53	No	No	No	Yes	No	No		874/07	lax LES					Yes	No	Yes	Yes
54	Yes	Yes	Yes	Yes	Yes	No		916/07				I		Yes	No	Yes	Yes
55	Yes	Yes	No	Yes	No	Yes		324/07		I	I			Yes	No	Yes	Yes
56	No	No	No	No	Yes	No		948/07	N					Yes	No	Yes	Yes
57	Yes	Yes	No	Yes	No	No	Htn	1675/07			I			Yes	No	Yes	Yes
58	No	No	No	Yes	Yes	No		1384/07	lax LES					Yes	No	Yes	Yes
59	No	Yes	No	Yes	Yes	No		2538/07	N					Yes	No	Yes	Yes
60	Yes	No	No	Yes	Yes	Yes		1720/07	N					Yes	No	Yes	Yes
61	No	Yes	Yes	Yes	Yes	No	Htn	1556/07	lax LES				Yes	Yes	No	Yes	Yes
62	No	No	No	No	No	No		1508/07	pale					Yes	No	Yes	Yes
63	No	Yes	Yes	Yes	No	No		1671/07			I			Yes	No	Yes	Yes
64	No	No	No	No	No	No		1669/07		N	I			Yes	No	Yes	Yes
65	Yes	Yes	Yes	Yes	Yes	Yes		828/06	OGJ polyp	N	I		Yes	Yes	No	Yes	Yes
66	Yes	No	No	Yes	No	No	Htn						Yes	Yes	No	Yes	Yes
67	No	No	Yes	Yes	No	No		1950/07			I			Yes	No	Yes	Yes
68	Yes	Yes	Yes	Yes	No	No		2726/07					Yes	Yes	No	Yes	Yes
69	No	No	No	Yes	Yes	No	Htn	1861/07	N					Yes	No	Yes	Yes
70	No	No	No	No	No	No	DM	57/08		U			Yes	Yes	No	Yes	Yes
71	No	No	No	No	No	No		2364/07	N								
72	No	No	No	No	No	No		6077/07			I			Yes	No	Yes	Yes
73	No	No	No	Yes	Yes	No		2518/07				U		Yes	No	Yes	Yes
74	No	No	No	No	No	No		349/04				U		Yes	No	Yes	Yes
75	No	No	No	Yes	No	No	DM,Htn							Yes	No	Yes	Yes
76	No	No	No	No	Yes	No		2086/07	N					Yes	No	Yes	Yes
77	No	No	No	Yes	No	No		2161/07			E			Yes	No	Yes	Yes
78	No	No	No	No	No	Yes		12/3/2007		I				Yes	No	Yes	Yes
79	No	Yes	No	Yes	No	No	Htn	3222/07	N					Yes	Yes	Yes	Yes
80	Yes	Yes	Yes	No	Yes	No		3341/07	lax LES	I				Yes	No	Yes	Yes
81	No	Yes	Yes	Yes	No	No		198/08		I				Yes	No	Yes	Yes
82	Yes	Yes	Yes	Yes	No	No		157/08		I				No	No	Yes	Yes
83	No	No	No	Yes	Yes	Yes	DM	421/08	N					Yes	Yes	Yes	Yes
84	Yes	Yes	Yes	Yes	No	No		582/08	N					Yes	No	Yes	Yes
85	No	No	No	Yes	No	No	Htn	317/08	N					Yes	No	Yes	Yes
86	No	No	No	Yes	No	Yes		556/08	N					Yes	No	Yes	Yes
87	Yes	No	No	Yes	No	No		927/08	N					Yes	No	Yes	Yes
88	No	Yes	Yes	No	Yes	No		653/08	N					Yes	No	Yes	Yes

Sl.No	hoarseness	chr cough	recLRI	caries	wt loss	UGI bleed	comorb	Scopy No	Normal	OeN/I/E/U	StN/I/E/U	DuN/I/E/U	HiatusH	Dietchange	L S mod	Resp to Rx	Rec
89	Yes	No	No	No	Yes	No		810/08	N					Yes	No	Yes	Yes
90	No	No	No	No	No	No		896/08	N					Yes	No	Yes	Yes
91	No	Yes	No	Yes	No	No		978/08	N					Yes	No	Yes	Yes
92	No	No	No	No	No	No		757/08				diverticul		Yes	No	Yes	Yes
93	No	No	No	No	Yes	No		1816/08	N					Yes	No	Yes	Yes
94	No	No	No	Yes	No	No		1277/08		I				Yes	No	Yes	Yes
95	Yes	No	No	No	No	Yes								Yes	Yes	Yes	Yes
96	Yes	No	No	No	No	No								Yes	No	Yes	Yes
97	No	No	No	Yes	Yes	No		1429/08	N					Yes	No	Yes	Yes
98	Yes	No	No	No	Yes	No		517/08		I				Yes	No	Yes	Yes
99	No	No	No	Yes	No	No	DM										
100	No	No	No	Yes	No	No	DM	1599/08		I,U,strictur			Yes	Yes	Yes	Yes	Yes
101	No	No	No	No	No	No		1791/08		I				Yes	Yes	Yes	Yes
102	No	No	No	No	Yes	No		44953				U		Yes	No	Yes	Yes
103	No	No	No	No	No	No		108/08		I	U perf	U		Yes	No	Yes	Yes
104	No	No	No	No	Yes	No	HIV?	1732/08		I-monilial				Yes	No	Yes	Yes
105	No	No	No	Yes	Yes	No				I	I		Yes	Yes	No	Yes	Yes
106	No	No	No	Yes	Yes	No				I				Yes	No	Yes	Yes